# CARDIOVASCULAR SURGERY


By

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THIS BOOK
IS HUMBLY DEDICATED
TO THE PATIENT

WHOM GOD HAS PLACED IN OUR HANDS TO DETERMINE AND TREAT HIS ILLS.
IF WE SUCCEED AND RELIEVE THE GREAT HEALER OF A PART OF HIS TASK
PERHAPS WE MAY JUSTIFY OUR EXISTENCE AND HIS FAITH IN OUR ABILITY

# PREFACE

The surgical profession has been interested in developmental defects lesions and diseases of the cardiovascular system as long as the art and science of surgery have been studied and practised. Many of the fundamental principles of this branch of surgery were postulated and even tried by our worthy predecessors in this field. The advancement and improvement in such ancillary fields of surgery as anesthesiology blood and fluid balance, antisepsis and the antiobiotic substances together with the anti thrombotic drugs has made our modern conception and treatment of many of these lesions possible. Thus the origin of the field of successful cardiovascular surgery on the human is within the memory of us all and dates back only fifteen years or so.

With our longevity increasing each year the treatment of cardiovascular diseases and their complications becomes most important. For the individual who has reached fifty years of age another twenty-three to twenty-eight years of life can be expected. Of this group six out of every ten will die of some cardiovascular lesion. Mathematically it would appear that we must be most interested in this group of diseases.

The great interest in this branch of surgery is understandable. If one loses the nerve muscle or bone control of a body segment that portion of the body may be functionless. If the blood supply to the area is lost however that region necessarily must die. If the part is essential to life as for example the heart the brain the kidney etc the person dies. Thus cardiovascular disease deals with the fundamental part of the life of the human himself.

One becomes humble in the study of the cardiovascular system. In our efforts to describe the methods of therapy available today we find that many times we are but mimics of greater thinkers and doers who preceded us. If we succeed where they failed it is only because we have gained from the bitter experiences of those whom we follow and the great Healer perhaps has placed better tools in our hands.

To deserve its publication a book must have a purpose. This book's purpose is to bring to surgeons internists and students a summary of accepted or acceptable treatment for cardiovascular lesions. These measures are in active use in a large vascular clinic and hospital. The author and his Clinic try to keep honestly patient-minded. By this is meant that we use the best methods of therapy that can be found of benefit to our patients. No effort is made to prove some measure superior to one already good. Each patient is considered similarly to the way he would be considered were he the father or mother of one of the doctors.

All the new reports are not included. It is accepted that tomorrow there may be a new discovery which makes some dogma of this text obsolete. It is hoped however that this book will bring the subject matter up to this date to the reader. It is also the hope that the context may act as a

post-graduate review to the physician who physically cannot take post-graduate courses to acquaint him with recent knowledge of this rapidly changing field It is the author's thought also that this book may aid in undergraduate teaching This subject in our busy medical schools is left to be covered in part by other required courses Finally, with our age of rapid travel, industrialization, and major wars which means more extensive and severe injuries, it is hoped that this book may help in a more rational treatment of wounds and injuries of this system

This book has been assembled in sections The first section deals with the new nomenclature standardization by the New York Heart Association and the case history and physical examination The second section discusses anesthesia, cardiac arrest and resuscitation, blood and fluid balance and the employment of antibiotic drugs The third section details the operations on the heart Cardiac physiology, thoracotomy and diagnostic measures are included for both congenital and acquired heart disease The treatment of coronary occlusion, tumors of the heart and lesions of the pericardium are covered Accepted and experimental methods are discussed. The arterial system is divided into the occlusive and spastic lesions, injuries to vessels, and the therapy for aneurysms and embolisms Recent advances in artery surgery and grafting and blood vessel banks are shown. The surgical treatment of hypertension is discussed

The venous system is presented in all its aspects from congenital anomalies to the complications of clotting In like manner, the changes in the therapy of lymphatic obstruction are shown A separate section deals with efforts to divert the circulation, methods of by-passing parts or sections of the heart and blood vessels, and other cardiovascular procedures such as angiography, cardioangiography, and cardiac catheterization The importance of skin grafting is stressed The surgical treatment of portal hypertension and the significance of atomic energy and radioactive isotopes are in the following section Carotid body tumor, carotid sinus syndrome, and a summary of the treatment of all vascular ulcers completes this section The final section discusses the importance of injuries and occupational hazards in the development of cardiovascular disease and in the patient who has such a disease

In a previous text devoted to the same subject on a more limited scale, I gave tribute to those to whom I am indebted for my training and surgical opportunities These included my mother and my family who sacrificed a great deal for me, my uncle, the late Dr Robert Emmett Farr of Minneapolis, who outlined my course and guided my early years, and, Dr W Wayne Babcock, who apprenticed me to him surgically and personally for four years and gave me liberally of his surgical knowledge Dr Charles G Heyd and the late Dr Thomas Russell of the New York Post-Graduate Hospital when that institution existed, and Dr Louis Rousselot and Dr John Mulholland in recent years gave their surgical aid, advice and faithful support Those in the cardiovascular field who have helped me unselfishly include Dr Irving S Wright, Dr A Wilbur Duryee, Dr Emile Holman,

Dr Teresa McGovern Dr George Humphries II Dr Alfred Blalock Dr Robert Gross Dr Charles Bailey and Dr John Madden These and Dr Willis Potts and Dr James White permitted reproduction of illustrations of their work The Surgeon General of the Navy the Bureau of Medicine and Surgery and the Naval Hospitals permitted reproduction of work performed on Naval personnel

In the preparation of this book my secretarial staff has not only been devoted but sacrificial in their aid These include Miss Nancy Brown, who worked loyally faithfully and unstintingly for innumerable hours Miss Betty Lee Gilman who continued her unselfish and excellent work with my first book and also arranged the bibliography and the many others who worked for a time My personal family were understanding and kind to a tired and harassed member Sister Loretto Bernard administrator and her Staff of the Sisters of Charity at St Vincent s Hospital of the City of New York have been most kind and cooperative Mother Alice and her Staff of the Sisters of the Third Order Regular of St Francis of Allegany New York at St Clare s Hospital of New York City have been gracious in their help Lea & Febiger and their staff were most helpful in the completion of this work

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# SECTION I

## Nomenclature, Case History, and Physical Examination *of the Patient*

## Chapter 1

## NOMENCLATURE OF DISEASES AND ABNORMALITIES OF THE CARDIOVASCULAR SYSTEM

THE American Heart Association has assumed the responsibility for coordinating the nomenclature of the diseases and abnormalities of the cardiovascular and lymph systems Such clarification of the names and terms and definition of what is meant by the names used to describe lesions is necessary as this field widens with the increased study and knowledge The first nomenclature was completed in 1940 A revision of it with modifications deletions and additions made necessary by modern knowledge and the changes incident to surgical interest was completed and published in 1953 The work of the Committee was painstaking and time consuming as differences of opinion had to be composed The author served on both of these Nomenclature Committees The modern one is incorporated in this book as it applies to the vascular diseases The Nomenclature of all of the subdivisions of cardiac disease is not included since many of its ramifications do not apply to surgery Those interested in the Cardiac Nomenclature or the subheads descriptions are referred to the Edition of *Nomenclature* Nomenclature and Criteria for Diagnosis of Diseases of the Heart published by the New York Heart Association Committee on Nomenclature

The Nomenclature used in this book for the lesions and diseases of the heart which will be discussed follows

1 Wounds of the Heart
2 Congenital Heart Lesions
   (*a*) Pulmonary Stenosis (The Tetralogy of Fallot)
      (1) Pure pulmonary stenosis
      (2) Infundibular *stenosis associated* with interventricular septal defect and overriding of the aorta
      (3) Variations of the above
   (*b*) Patent Ductus Arteriosus
   (*c*) Coarctation of the Aorta
   (*d*) Anomalies of the Arch of the Aorta and its Branches
   (*e*) Anomalies of the Pulmonary Vessels
   (*f*) Transposition of the *Aorta* and Pulmonary Artery

(*g*) Septal Defects
  (1) Interatrial Defects
  (2) Interventricular Defects
  (3) Other Congenital Defects

3 Acquired Heart Lesions
  (*a*) Mitral Stenosis
  (*b*) Mitral Insufficiency
  (*c*) Aortic Stenosis
  (*d*) Aortic Insufficiency
  (*e*) Tricuspid Stenosis
  (*f*) Tricuspid Insufficiency
  (*g*) Various Combinations of *a*, *b*, *c*, *d*, *e*, *f*
  (*h*) Congestive Failure associated with above lesions

4 Coronary Occlusion

5 Tumors of the Heart

6 Pulmonary Embolism

7 Lesions of the Pericardium
  (*a*) Pericarditis
  (*b*) Pericardial tumors

It is obvious that the above Nomenclature does not follow that approved by the American Heart Association For our purposes, however, it is more workable in discussing lesions of the heart amenable to surgery

## NOMENCLATURE FOR DIAGNOSIS OF VASCULAR DISEASES

### DISEASES OF ARTERIES AND ARTERIOLES

#### Functional Conditions (Vasomotor)

Key to Standard Nomenclature uses various numbers in the 46 and 47 series to indicate particular arteries and 47x to indicate arterioles in general

a with vasospastic manifestation

b with vasodilator manifestation

* This number is used in Standard Nomenclature

† If the cause of the vascular disease is a disease of another tissue, the primary disease should be listed by its own anatomical and etiological numbers, *e g*, cervical rib 229-031

° Indicate affected region of body by first three digits

#### A Vasoconstrictor

| | |
|---|---|
| 1 Raynaud's syndrome (primary Raynaud's Disease) | 47 —582 |
| 2 Raynaud's syndrome (secondary) | |
| a Traumatic vasospastic syndrome | 47 —432a |
| b Neurovascular mechanisms | |
| (1) Cervical rib† | 47 —031a |
| (2) Scalenus anticus syndrome† | 47 —131a |
| (3) Hyperabduction syndrome† | 47 —131a |
| (4) Spondylitis† | 47 —635a |
| (5) Neuritis† | 47 —586a |

| | |
|---|---|
| c Secondary to organic vascular disease | |
| (1) Arteriosclerosis† | 47 —516a |
| (2) Thrombo-angiitis obliterans† | 47 —51xa |
| (3) Syphilitic arteritis† | 47 —51xa |
| d Secondary to Intoxications | |
| (1) Nicotine | 47 —309a |
| (2) Tobacco | 47 —360a |
| (3) Arsenic | 47 —3114a |
| (4) Ergot | 47 —307a |
| (5) Lead | 47 —3112a |
| e Scleroderma | 47 —071a<br>114—071* |
| f Miscellaneous mechanisms | 47 — a |
| 3 Acrocyanosis | 47 —518a<br>098—518* |
| 4 Cutis marmorata | 47 —586a<br>11 —440* |
| 5 Vasospasm secondary to | |
| a Lesions of peripheral nerves† | 47 —586a |
| b Lesion of brain and spinal cord† | 47 —586a |
| c Thrombophlebitis† | 47 —586a |
| d Embolism† | 47 —406 4a |
| e Thrombosis† | 47 — 7a |
| f Trauma (post traumatic reflex sympathetic dystrophy Sudeck's atrophy post traumatic osteoporosis) | 47 —4 a<br>2 —400 0* |

B VASODILATOR

| | |
|---|---|
| 6 Erythermalgia primary | 47 —581b |
| 7 Erythermalgia secondary to | |
| a Polycythemia vera† | 47 —541b |
| b Arteriosclerosis† | 47 —516b |
| c Thrombo-angiitis obliterans† | 47 —515b |
| d Trauma | 47 —4 b |
| e Miscellaneous factor | 47 — b |

ORGANIC CONDITIONS (STRUCTURAL)

A. OCCLUSIVE (organic)

| | |
|---|---|
| 8 Arteriosclerosis | |
| a Atherosclerosis obliterans | 46 —952 4 |
| b Medial (Mönckeberg's) arteriosclerosis | 4602—955 |
| c Combined | 46 —950 |
| 9 Thrombo-angiitis obliterans | 402—930* |
| 10 Essential polyangiitis (periarteritis nodosa) | 402—931 4 |
| 11 Cranial arteritis (temporal arteritis) | 474—931 4 |
| 12 Ergotism | 47 —307 4 |
| 13 Arteritis secondary to | |
| a Infectious diseases† | 47 —1 4 |
| b Local inflammatory processes† | 47 —1 4 |
| 14 Hypertensive vascular disease | 460—533 |

| | | |
|---|---|---|
| 15 | Arteriolitis, secondary to | 47x—1 4 |
| | a Infectious diseases† | —1x0 4 |
| | b Local inflammatory processes† | 47x—1 4 |
| | c Lupus erythematosus disseminatus† | 47x—910 4 |
| | d Idiopathic arteriolitis | 47x—930 4 |
| 16 | Arterial Thrombosis | |
| | a Associated with infectious diseases† | 46 —1 7 |
| | b Associated with blood dyscrasias† | 46 —54 7 |
| | c Secondary to trauma or compression† | 46 —4 7 |
| | d Secondary to surgery† | 46 —415 7 |
| | e Associated with parturition† | 46 —417 7 |
| | f Associated with cardiac insufficiency† | 46 —519 7 |
| | g Associated with slowed blood stream† | 46 —510 7 |
| | h Associated with exposure to radiation | 46 —47 7 |
| | i Idiopathic | 46 —619 |
| 17 | Abscess of wall of artery | 46 —1 2 |
| 18 | Frost-bite | —448*° |
| | | 47 —448 |
| 19 | Pernio | 110—446*° |
| | | 47x—446 |
| 20 | Livedo reticularis | 47x—900a |
| | | 147—5x2*° |
| 21 | Arterial embolism | |
| | a Thrombus† | 46 —496 4 |
| | b Fat† | 46 —426 |
| | c Air† | 46 —427 |
| | d Bacterial† | 46 — 4 |
| | e Neoplastic† | 46 —8 4 |
| | f Fungus† | 46 —2 4 |
| | g Inorganic substances | 46 —429 4 |

B Non-occlusive (organic)

| | | |
|---|---|---|
| 22 | Aneurysm | |
| | a Congenital | 46 —015 |
| | b Syphilitic | 46 —147 6 |
| | c Arteriosclerotic | 46 —942 6 |
| | d Mycotic | 46 —100 6 |
| | e Traumatic | 46 —4 6 |
| | f Embolic† | 46 —618 6 |
| | g Idiopathic | 46 —910 6 |
| 23 | Essential polyangiitis (periarteritis nodosa) | 402—931 |
| 24 | Arteriovenous anastomosis (fistula) | |
| | a Congenital | 402—029 |
| | b Traumatic | 402—4 3 |
| | c Secondary to malignancy | 402—8 3 |
| | d Secondary to bacterial infections | 402—1 3 |
| | e Secondary to fungus infections | 402—2 3 |
| 25 | Congenital anomalies of artery | 46 —0 |
| 26 | Trauma of artery | 46 —4 |
| 27. | Scalenus anticus syndrome† | 46 —434 |
| 28 | Rupture of artery† | 3 —1 5 |
| | | —1 7 |
| 29 | Effects of expos[illegible] | 47 |

## DISEASES OF VEINS

### Functional Conditions (Vasomotor)

| | Code |
|---|---|
| Spasm | 48 —582<br>—4 a |

### Organic Conditions (Structural)

#### A Occlusive

| | Code |
|---|---|
| 1 Thrombophlebitis and venous thrombosis (phlebothrombosis) | |
| a Primary | |
| (1) Thrombo-angiitis obliterans | 402—930* |
| (2) Migratory thrombophlebitis | 480—930 7 |
| (3) Essential or idiopathic, local | 48 —000 7 |
| b Secondary to | |
| (1) Mechanical injury | 48 —4 7 |
| (2) Muscular effort or strain | 48 —43x 7 |
| (3) Chemical injury | 48 —3 7 |
| (4) Inflammatory or suppurative lesions (etiologic agent to be indicated) | 48 —1 7<br>—2 7 |
| (5) Infectious diseases† | 48 —1 7 |
| (6) Severe ischemia† | 48 —514 7 |
| (7) Varices† | 48 —522 7 |
| (8) Blood dyscrasias | |
| (a) Polycythemia† | 48 —541 7 |
| (b) Myelogenous leukemia† | 48 —543 7 |
| (c) Lymphatic leukemia† | 48 —545 7 |
| (d) Pernicious anemia† | 48 —542 7 |
| (e) Disturbances of blood-clotting mechanism† | 48 —540 7<br>48 —549 7 |
| (f) Other blood dyscrasias† | 48 —5 7 |
| (9) Cardiac insufficiency† | 48 —510 7 404 |
| (10) Carcinoma† | 48 —8 7 |
| 2 Neoplastic invasion of vein | 48 —8 4 or 7 |
| 3 Venous compression by | |
| a Gravid uterus† | 48 —435 4 |
| b Neoplasm† | 48 —430 4 |
| c Aneurysm† | 48 —435 4 |
| d Scar tissue† | 48 —435 4 |
| e Scalenus anticus syndrome† | 48 —434 4 |
| f Hyperabduction syndrome† | 48 —431 4 |
| g Fractures† | 48 —436 4 |
| h Dislocations† | 48 —434 4 |
| i Increased intra-abdominal pressure† (ascites) | 48 —522 4 |

#### B Non-occlusive

| | Code |
|---|---|
| 4. Varicose veins | |
| a Primary | 48 —9x0 |
| b Secondary to | |
| (1) Posture | 48 —431 9 |
| (2) Occupation | 48 —432 9 |
| (3) Clothing | 48 —433 9 |
| (4) Proximal obstructive lesions or pressure† (See II A, 3-Diseases of Veins) | 48 —522 9 |
| (5) Thrombophlebitis† | 48 —522 9 |

| | | |
|---|---|---|
| | (6) Arteriovenous anastomosis† | 48 —522 9 |
| | (7) Hemangioma | 48 —850 |
| | (8) Congenital anomalies of veins | 48 —010 9 |
| 5 | Arteriovenous anastomosis (fistula) | |
| | a Congenital | 402—029 |
| | b Traumatic | 402—400 3 |
| | c Secondary to malignant lesions† | 402—8 3 |
| | d Secondary to bacterial infections† | 402—1 3 |
| | e Secondary to fungus infections† | 402—2 3 |
| 6 | Aberrant position of vein | 48 —021 |
| 7 | Hypoplasia of vein | 48 —016 |
| 8 | Phlebectasia | 48 —015 |
| 9 | Periphlebitis | 48 —190 |
| 10 | Phlebosclerosis | 48 —952 |
| 11 | Rupture of vein | 48 —4 5<br>—1 5 |

## NEOPLASMS OF BLOOD VESSELS

### Hemangioma

| | | |
|---|---|---|
| 1 | Cavernous hemangioma | 490—850A |
| 2 | Capillary hemangioma | 490—850A |
| 3 | Plexiform hemangioma | 490—850A |
| 4 | Sclerosing hemangioma | 490—858A |
| 5 | Syndromes with hemangiomas | |
| | a Multiple hemangiomas and chondromas (Kast's syndrome) | 490—850A<br>—873B |
| | b Hemangioma of retina and central nervous system | x23—850A<br>900—850A |

### Hemangio-Endothelioma

| | | |
|---|---|---|
| 6 | Hemangio-endothelioma—benign | 490—850A |
| 7 | Hemangio-endothelioma—malignant | 490—850B |

### Sarcoma

| | | |
|---|---|---|
| 8 | Angiosarcoma | 490—850G |
| 9 | Kaposi's sarcoma | 490—852 |
| 10 | Ewing's sarcoma | 490—875G |
| 11 | Glomus tumor (angioneuromyoma) | 490—8532 |
| 12 | Hemangiopericytoma | 490—8531 |
| 13 | Telangiectasis | |
| | a Hereditary hemorrhagic telangiectasia | 490—851A |
| | b Papillary varices | 11[illegible]—8024 |
| | c Spider angioma | 110—850A* |

## DISEASES OF THE LYMPHATIC SYSTEM

| | | |
|---|---|---|
| 1 | Lymphangioma | 540— |
| | a Simplex | 540— |
| | b Cysticum | 540— |
| 2 | Lymphangiosarcoma | 540 |
| 3 | Lymphedema | |
| | a Primary | |
| | (1) Congenital (Milroy's disease) | [illegible] |
| | (2) Praecox | [illegible] |

| | |
|---|---|
| b Secondary to | |
| (1) Surgical removal of lymph nodes | 55 —415 8 |
| (2) Neoplastic invasion of lymph nodes | 55 —8 8 |
| (3) Lymphadenitis due to | |
| (a) X ray | 55 —4711 8 |
| (b) Pyogenic infection | 55 —1 8 |
| (c) Granulomatous infections (etiologic agent to be indicated) | 55 —2 8<br>—1 8 |
| (4) Dependency edema | 540 —431 8 |
| 4 Inflammatory lesions | |
| a Acute lymphangitis | 54 —1 |
| b Chronic lymphangitis | 54 —1 |

## DISEASES OF MINUTE VESSELS

### Increased Fragility of Vessels

| | |
|---|---|
| 1 Infectious purpura | |
| a Bacterial | 400—1 5 |
| b Virus and other micro-organisms | 400—1 5 |
| 2 Toxic purpura | |
| a Arsenic | 400—3114 5 |
| b Phosphorus | 400—3123 5 |
| c Phenolphthalein | 400—34211 5 |
| d Heparin and related substances | 400—3829 5 |
| e Coumarin derivatives and related substances | 400—300 5 |
| f Venom | 400—381 5 |
| 3 Purpura due to avitaminosis | |
| a Scurvy† | 400—763 5 |
| b Lack of vitamin K† | 400—766 5 |
| c Other vitamin deficiency† | 400—7 5 |
| 4 Purpura secondary to increased venous pressure | 400—522 5 |
| 5 Menstrual purpura† | 490—786 5 |
| 6 Senile purpura | 400—707 5 |
| 7 Idiopathic purpura | *507—7911 |
| a Henoch's purpura | 400—9x7 |
| b Schoenlein's purpura | 490—9x71 |
| 8 Allergic purpura | 400—390 5 |

### Increased Permeability of Vessels

| | |
|---|---|
| 9 Urticaria | 490—390 8 |
| 10 Sensitivity to physical agents | |
| a Mechanical | 490—4 8 |
| b Cold | 400—44x 8 |
| c Heat | 400—440 8 |
| 11 Hematogenic purpura | |
| a Thrombocytopenia† | 490—548 5 |
| b Leukemia† | 400—543 5<br>—545 5 |
| c Aplastic anemia† | 400—542 5 |
| d Granulocytopenia† | 400—544 5 |
| e Disturbances of clotting mechanism† | 490—549 5 |
| 12 Local inflammation† | 490—1 8 |

| | | |
|---|---|---|
| 13 | Anaphylactic shock† | 490—390 S |
| 14 | Traumatic shock† | 490—591 S |
| 15 | Burns† | 490—441 S |
| 16 | Frost-bite† | 490—448 S |

As far as it is possible the form and order of the nomenclature is followed Such changes as are made were necessary when the surgical part of the diagnosis or treatment deviates from the standard

The Nomenclature Committee was.

Irving S Wright, *Chairman*
William T Foley, *Secretary*

Nelson W Barker
Joseph E Flynn
Paul Klemperer
Gerald H. Pratt
Wallace M Yater

Many of the diseases listed can now be considered entities The Committee recognized, however, that any nomenclature must be subject to revision The hope of the Nomenclature Committee and its parent organization, The American Heart Association, is that authors, research workers and others interested in cardiovascular diseases will use the preferred terms In this way a universal terminology will develop and all will know what is meant by a described lesion, not only in this country but throughout the world The first section of this book describes the scope of cardiovascular diseases and such fundamental problems as the importance of a history and a complete physical examination A large section is devoted to diagnosis and surgical treatment of congenital and acquired cardiac lesions The remainder of the book is divided into sections corresponding to the various circulatory media, the arterial, the venous and lymphatic systems Another section contains the description and treatment of such specialized diseases as portal hypertension, ulcers due to various causes, and the relationship to trauma and cardiovascular diseases, particularly in their medical-legal sense The technic of delineation of the cardiovascular system with radio-opaque due and the roentgen interpretation of such pictures, as well as the use of radioactive isotopes in the diagnosis and treatment of these lesions is included The importance of injuries to the heart and vessels, which increases with more rapid and distant travel and with the greater destructiveness of modern warfare, requires special discussion The Table of Contents describes the sections and the chapters

Chapter

2

# THE HISTORY THE PHYSICAL EXAMINATION AND THE LABORATORY STUDY OF A PATIENT

Of all the diagnostic measures available to us in medicine and surgery none is equal in value to an accurate history and complete physical examination of the patient These are our prime tools with which we will fashion the diagnosis They are as valuable and important to us as are addition and subtraction in solving a mathematical problem In many ways the similarity exists The history and examination of the patient add important and subtract irrelevant facts about our patient s problem Our aim is to provide the correct answer As in arithmetic there is only one correct answer

## A THE HISTORY

The patient should be allowed to talk. He should be asked why he is consulting the doctor He should then be permitted to tell his story in his own words the only direction from the doctor being towards chronological order After notes have been made direct questions can be asked to clarify obscure data Specific questions necessary for differential diagnosis should be clear Clear answers should be obtained but the patient should not be led in such questions Members of the family should contribute facts after the patient himself has completed his story It is important for the doctor to go over each point in this history specifically The history from the referring doctor may or may not contribute to the final analysis

The questions should be simple For example when a patient is asked how far he can walk he should be told this means on level ground at his average walking rate The reaction to rest and the time interval before walking can be resumed is a good indication of the extent of claudication The previous state of health the medical and surgical therapy and the family history personal habits weight change occupation as well as menstrual and obstetrical history is of value One must recognize attempts to enlarge or minimize symptoms Family or personal reticence should be recognized as such The importance of the history taking must be explained to the patient. Most of the time the patient will tell the doctor the diagnosis in lay terms if he will listen A history chart which has served as a good guide at our clinic is incorporated on pages 21–23

## B. PHYSICAL EXAMINATION

The patient should be told that a complete physical examination is essential. When the patient reaches a specialist he often expects to have only the part with which he is concerned examined. Previous physical examinations even if multiple and by competent physicians should not be accepted in lieu of the surgeon's own examination. Such an examination gives the surgeon a sense of security in his diagnosis which cannot be obtained in any other way. The therapy he will suggest may vary dependent upon the general physical status and should not be evaluated until the physical examination is complete. An unsuspected and serious lesion may be found. It may be that the patient's complaints are of symptoms of an underlying and more serious lesion. For example, phlebitis is a frequent complication of cancer. By complete examination is meant the use of the examiner's five senses. This includes an examination of all of the apertures of the body, including the vagina and rectum. Speculum and proctoscopic examinations and the use of other specialized media such as the ophthalmoscope are necessary.

**Specific Tests.**—*Ophthalmoscopic Examination.*—The status of the arteries and veins can be seen clearly in the fundus of the eye. The diagnosis of some intracranial lesions can be confirmed in this way. A comparison of the patient's blood vessels and his age may give important prognostic and therapeutic data.

*Palpation and Auscultation.*—All the vessels in the body should be felt and listened to with a stethoscope. The presence or absence of pulsations, thrills and bruits is important. The character of the vessel can be determined by its consistency.

*Blood Pressure.*—The systolic and diastolic reading should be recorded in all four extremities. These readings should be taken in the lying, sitting, standing positions and the response to effort noted. This is important in the diagnosis of hypertension, cardiac lesions, aneurysms, coarctation of the aorta, etc.

*Oscillometric Readings.*—These readings should be registered in all extremities unless constriction of the vessels of an extremity might be deleterious to the circulation. In the lower extremity the readings in the foot are most important. This requires the use of an apparatus sensitive enough to register such readings. In many diseases the peripheral circulation is the one that fails first.

## C. LABORATORY EXAMINATIONS

A good physical examination today should include a complete blood count, a urinanalysis, a sedimentation rate and a test of the important chemicals of the blood. A chest *x*-ray and electrocardiograph, angiography, and tests of the basal metabolism and vitamin supply depend upon the lesion suspected. Temperature of the body as well as the skin can be determined by thermocouple readings. These should be performed if possible in a thermostatically-controlled atmosphere. If this is not available, a relative test can be done by comparing the affected and unaffected

THE ST VINCENT S HOSPITAL OF THE CITY OF NEW YORK

VASCULAR CLINIC

Clinic No. ——
Date ——

NAME | [illegible] | AGE

[illegible] | SEX | BIRTHPLACE

[illegible] | DATE

CLASSIFICATION [illegible]

DIAGNOSIS
(not to be filled in)

I CHIEF COMPLAINT (Reason for being patient)

II. PRESENT ILLNESS (Specify a. duration; b. precipitating factors like exposure, injury etc; c. progression; d previous therapy and response)

[illegible]

Activity Pain,
[illegible]
[illegible]
[illegible]
Location——
Severity——
[illegible]——
[illegible]——
Relieved by——
Rest Pain:
Duration,
Location——
Severity——
[illegible]——
Relieved by——

Color Changes in Extremities
At Cardiac Level——
On Elevation
On Dependency
Temperature Changes
Response to Sensitivity
Heat——
Cold——
Trophic Changes,
[illegible]
[illegible]
Type——
Location——
[illegible]——
Previous Therapy——

[illegible]

[illegible]
Duration——
Location——
Previous Therapy Response
[illegible]
Location——
Duration——
[illegible]——
Complications——

[illegible]
Duration——
Location——
[illegible]——
[illegible]——
Skin Changes
Other

[illegible]

[illegible]
Type——
Duration——
Therapy——
Response——

[illegible]
Type——
Duration——
Therapy——
[illegible]——
Other



II PERSONAL HISTORY

Occupation, past and present

Use of—
Tobacco (duration, kind and amount)

b. Alcohol (duration, kind and amount)

c. Rye Bread (duration, kind and amount)

d. Drugs (reason, ergot, kind, etc.)

Exposure to elements

Family history of vascular or other disease

Pediatric history

III. PAST HISTORY

Diseases (vascular, cardiac, diabetes or other metabolic dysfunctions, infections, allergies, etc.)

Operations

Fractures or Injuries

Pregnancies

Venereal disease

Variations in weight

IV GENERAL SYMPTOMATOLOGY

V GENERAL PHYSICAL EXAMINATION

Weight
B P R——
L——

VI ORTHOPEDIC STATUS:

ST VINCENT'S HOSPITAL

Vascular Clinic

Chart of Oscillometric Readings

Name Chart Number

Age Sex Blood Pressure Date

14 12 10 8 7 6 5 4 3 2 1

200 180 160 140 120 100 80 60 40 20

Right Lower Extremity

14 12 10 8 7 6 5 4 3 2 1

200 180 160 140 120 100 80 60 40 20

Left Lower Extremity

Legend—Foot ————————

Above ankle . . . . . . . .

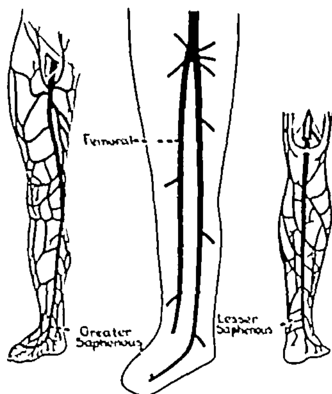
Below knee x x x x x x x x

Above knee o o o o o o o o

## ST. VINCENT'S HOSPITAL

[illegible] Vascular Clinic [illegible]

Name ______________ Date ____________ Clinic No ______




Indicate blowout points ulcerations etc on center diagra n

## ST. VINCENT'S HOSPITAL

[illegible] CLINIC

Name ______________ Date ______________

Patient No. ______________

[illegible] TEMPERATURE READINGS

| Time | [illegible] | [illegible] | | | | | | |
|---|---|---|---|---|---|---|---|---|
| RIGHT LEG<br>ARM | | | | | | | | |
| LEFT LEG<br>ARM | | | | | | | | |

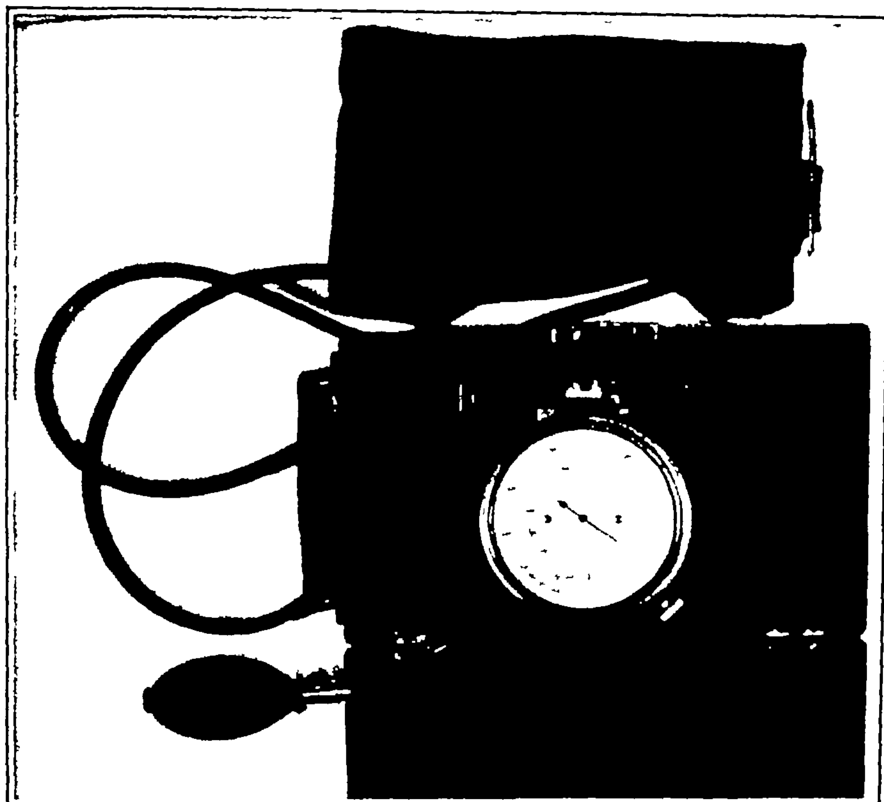


Fig. 1.—Von Recklinghausen Oscillometer.

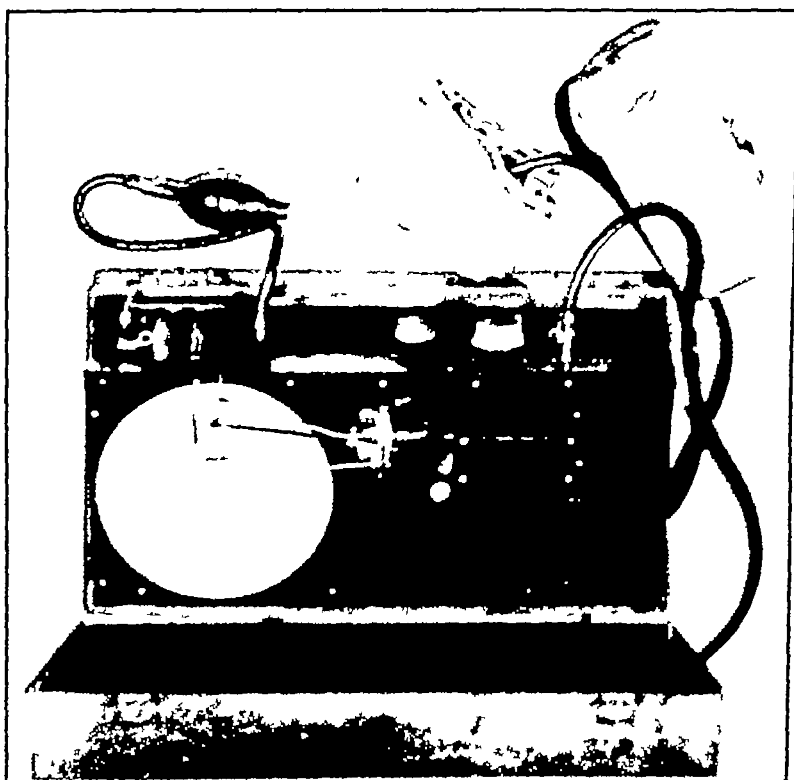


Fig. 2.—Tycos Recording Oscillometer.

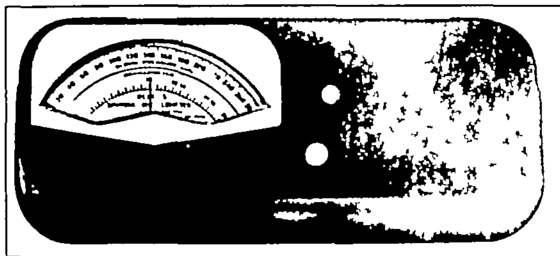


Fig. 3.—Collins Oscillometer.

part. Capillary microscopy, oxygen saturation, angiocardiography and cardiovascular catheterizations should be used only when needed to augment the clinical opinion.

**The Cardiac Patient.**—An evaluation of the cardiac patient and his possible selection for operation should be done in a special clinic by a group selected for their knowledge and experience in both medicine and surgery as it pertains to the heart. The internist should be a specialist in both internal medicine and cardiology. The children's specialist requires both pediatric and pediatric cardiology qualifications. The roentgenologist must be expert in interpreting the shadows and spaces so essential for diagnosis. In the past, the ability to make diagnosis was based on the simple tests whenever possible. To this group must be attached someone well trained in cardio-pulmonary physiology. Presumably such a member of the team will have angiocardiographic and catheterization training. It is important to preform pressure and oxygen determinations in questionable cases. In a few patients catheterization is required. It is important not to perform additional tests when they are not required. Such tests, even in the hands of experts, have caused fatal arrhythmias.

The requirements for making a diagnosis and determining the operability of the patient are detailed in the chapters on cardiac disease and therefore are not repeated here. The ultraconservative mind must share with the over-anxious surgeon responsibility for deaths through non-selection or poor selection of patients for the operation. In this field the surgeon must be more than an excellent technician. He should remember, however, that most of the theory and physiology of modern cardiac surgery was developed by a physiologist and internist. The ideal cardiac surgeon knows the stethoscope and electrocardiograph and most of all the symptomatology as well as he knows the scalpel and needle holder.

# SECTION II

## Ancillary Surgical Procedures

*Anesthesia, Blood and Fluid Balance, Cardiac Arrest and Resuscitation, Antibiotic Therapy*

The mortality of surgical operations has decreased. Operations which were not considered feasible or possible fifteen years ago now are performed successfully and safely. This change has been accomplished by the development of anesthesiology technics to their present high level and by the improvement in the use of the auxiliary surgical measures. These measures will be discussed in this chapter under the headings outlined above.

# Chapter 3

## ANESTHESIA

*For the Patient with Cardiovascular Disease and for Operations on the Cardiovascular System*

The anesthesia problem as it pertains to cardiovascular disease is divided into the care of the patient who has a cardiovascular lesion and must undergo some general surgical operation and the anesthesia which will be administered to the patient who is to undergo treatment for some cardiovascular lesion.

Collins[21,23] summarized the importance of the anesthesiologist. He must be a medical diagnostician cognizant of each surgical maneuver, a pharmacologist and a physiologist. Lastly, and probably of least importance, he must be able technically to anesthetize the patient with the minimal amount of the agent used. To this we add that he must know the art as well as the science of medicine.

The selection of the proper anesthesia for surgical procedures has reduced morbidity and mortality. This factor alone has placed many more operations within the scope of the surgeon. Indeed, modern anesthesiology with antibiotics and blood replacement have opened surgical vistas previously unknown. One has but to recall the apprehension and sleepless nights that followed the accidental opening of the pleura to realize the changed surgical picture today. Open resection of the colon is now routine. A few years

ago such surgery would not have been tolerated in any operating room. The surgeon thus had to acquire greater technical skill to perform an aseptic anastomosis. The surgical trainee should be reminded of these facts from time to time. It is not impossible that immunity to the antibiotic substances may be developed. The qualitative and quantitative replacement of body fluids is another historic forward stride. In the days of direct transfusion, such an event was an all day procedure. Now with the blood banks, the weighing of blood sponges and the oximeters the patient is maintained in his preoperative fluid and blood balance throughout serious operations, even those in which hemorrhage is severe. The anesthesia selection and administration is the third leg of our present surgical pedestal. Abdominal incisions may be extended into the thorax. One or both pleuræ may be entered. The pericardium can be opened. All this is possible with the controlled aeration of intratracheal anesthesia.

In operations on patients with cardiovascular diseases this selection of anesthetic agents becomes even more important. These patients have lesions of their basic circulatory system. It is certain that if one part of this system is affected adversely the rest of its components will be altered to some degree if the lesion exists sufficiently long. Thus, the patient with arteriosclerosis who needs an amputation has coronary occlusion potentialities. He is also a candidate for cerebral hemorrhage. His renal vessels may place him in pre-uremia status. In like manner, the patient with an arteriovenous fistula has cardiac enlargement and an increased blood volume. The patient with pathologic veins may develop a pulmonary embolism. The individual with a congenital or acquired cardiac defect presents special anesthesia problems.

A trained anesthesiologist must cope with all of these possibilities. Since there are not sufficient experienced men in this field, the selection of anesthesia, the addition of auxilliary drugs, and the recognition and therapy of anesthetic complications often become problems for the surgeon. No effort will be made to discuss the physiology of anesthesia since excellent treatises[21] on this subject exist. The discussion of the selection of anesthesias listed in this chapter is based on clinical experience. It is derived also from discussions with anesthesiologists. Differences of opinion may exist, but the basic anesthesia problems have been resolved. The discussion of the anesthesia problem has two facets. One is the patient with cardiovascular disease who must undergo a general surgical procedure. With increased longevity, more patients reach the age where a surgical operation is required for some disease associated with an older age. The second is the selection of an anesthesia for an operation on the cardiovascular system itself. The selection of the correct anesthetic agent should be individualized in each instance. Insistence on one type of anesthesia for all patients will result in complications. Thus an elderly individual can tolerate a controlled level spinal anesthesia for an amputation or sympathectomy where a general anesthetic would produce complications which might be fatal. In general the less alteration from the normal which can be achieved in anesthesia will be withstood the best. Depressing drugs and routine intravenous injections in patients with faltering circulatory systems will be reflected in an increased mortality and morbidity.

## GENERAL SURGICAL OPERATIONS ON PATIENTS WITH CARDIOVASCULAR DISEASE

Since many patients with occlusive arterial disease are reaching the older age levels they will become subject to the diseases and infirmities of this age, i e, carcinoma etc These patients will require surgical operations The anesthesia of choice for the lower extremities and abdomen will remain spinal anesthesia For a prolonged procedure this can be given continuously through a catheter introduced through the needle after which the needle is withdrawn For operations on the head and neck a local infiltration or field block perhaps is the safest agent For the thorax or the abdomen and thorax an endotracheal technic becomes obligatory Attention should be directed to minimizing the strain of fighting or choking during the introduction of the tube because the deficient cardiac reserve and the brittleness of the blood vessels in general will not tolerate it

Since we know now that occlusive arterial changes begin in nearly half of the patients after forty, this possibility should be considered in all patients over that age The heavy smoker is an added risk Intravenous anesthesia is dangerous in these patients but at times is necessary The anesthesiologist should keep the dosage of the pentathol minimal In this group of patients over-sedation and over-anesthesia both leading to hypoxia is the great danger The anesthesiologist must realize that the anesthesia is being given for the benefit of the patient and not so that the administrator will not be bothered for an hour or so The surgeon must sacrifice the optimum relaxation which makes his work simpler to keep the amount of anesthesia minimal in the interests of the patient s general welfare

**The Use of Adjunct Drugs** —Many new drugs are available to the anesthesiologist Curare and curare-like drugs add to the muscle relaxation There are innumerable sedative and soporific drugs There are vasodilators and vasoconstrictors In addition drugs are available to eliminate respiratory secretions and to reduce or remove the cardiac arrhythmias The modern anesthesiologist has a drug store at hand His function is to keep the patient comfortable and to prevent complications by avoiding them In the hands of the trained conservative anesthesiologist these drugs are safe In inexperienced hands however they may be dangerous and complicate the picture sometimes beyond correction A standardized anesthesia technic, without the introduction of innumerable medical additions is the safest course Valuable time may be lost attempting to determine whether the adverse status in such a patient is due to too much anesthesia surgery or the addition of some drug to which the patient s reaction is abnormal Again, the experience of the anesthesiologist is important and the cooperation of the anesthesiologist with the surgeon is a proven requirement for surgical success

**Anesthesia in the Operations for Occlusive Arterial Diseases** —

*Amputation* —An amputation to the patient is not unlike an execution He has been psychically shocked by the thought of the loss of a limb and possible helplessness The operation should proceed rapidly and quietly

The anesthesiologist should cooperate by having all of his "props" ready and avoiding delay. In most cases the patient should be operated upon and on his way back to his room in fifteen minutes. A one-leg spinal anesthesia has been adopted in preference to all other types of anesthesia for amputation. The drug can be "dripped" intradurally and placed at the proper level. Hypotension, shock, high levels and the other complications previously encountered can be eliminated completely by this technic. Any psychic reaction to the use of the saw can be assuaged by cotton or fingers in the ears and distraction. In the extremely obese patient the sitting position in which the patient's feet are at the same level as his hips facilitates the intradural injection (see Fig. 5). The anesthetic agent depends upon the length of time the operation will consume. Simple procaine (4.0 per cent) in doses of 75 mg. is sufficient for most such operations.

General anesthesia increases the complications both from the pulmonary and embolic points of view. The deep respiration essential to a profound anesthesia has resulted in aspiration of venous clots from pathologic veins in the leg. Atelectasis and pneumonia are other frequent complications.

*Local anesthesia* has been replaced in amputation for vascular diseases because of the constriction of small vessels which follows its infiltration. An anesthesia such as ethyl chloride likewise is contraindicated.

*Refrigeration anesthesia* was used for these amputations for several years before the modern spinal technic was available. The application of the ice technic produces an anesthesia, although the major nerves must be locally anesthetized with procaine prior to their division. The indication for this type of anesthesia is now remote. In a debilitated patient with severe sepsis, disorientation or out of diabetic control the application of the ice and subsequent amputation may be lifesaving. The ice blanket has replaced the old "ice box." If general anesthesia must be used, cyclopropane is preferred due to the higher percentage of oxygen which can be given with it.

**Angiospastic Diseases.**—Most of the operations on these patients are to interrupt the sympathetic system. For the lower extremities spinal anesthesia is the ideal one for lumbar sympathectomy. In the upper extremity thoracic sympathectomy requires endotracheal anesthesia, and cyclopropane has worked satisfactorily. The occasional amputation of a digit or a part rarely is a problem. Local anesthesia is again contraindicated because it contributes to the spasm. Intravenous anesthesia usually is contraindicated. It is wise to avoid introducing solutions into a patient's vein whether the artery or the vein is the primary site of the disease. The introduction of a needle into a vein may cause reflex spasm. An infiltration or a hematoma may complicate the vascular problem in the extremity. In the course of the patient's disease progression is to be expected. The patient may associate in his mind such disease advances with the "needle in the blood vessel."

**Anesthesia in Operations for Aneurysms, Arterial or Aortic Thromboses**

extremities local infiltration or block is safe and satisfactory For aneurysms of the heart or thoracic aorta a closed intratracheal type is mandatory Aneurysms of the abdominal aorta are nearly all of the arteriosclerotic type Spinal anesthesia given by the continuous drip method is the best one in our experience General anesthesia is dangerous We have observed the actual enlargement of the aneurysm on the operating table with strain during a difficult general anesthesia

Where the artery or aorta is to be opened for thrombectomy and endarterectomy the relaxation of the spinal anesthesia again contributes to the safety of the operation

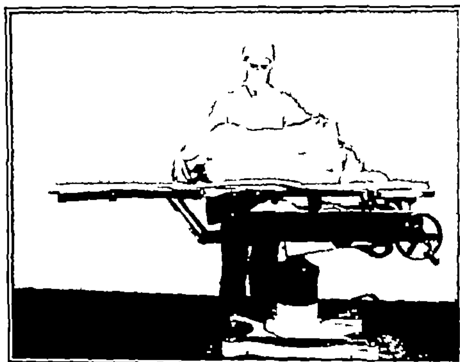


FIG 4 —Position of patient for spinal anesthesia.
(Collins Anesthesiology Lea & Febiger )

**Anesthesia in Embolism Operations** —For embolism of the aorta iliac artery or femoral artery local anesthesia is satisfactory The vessel is opened just below and/or just above the inguinal ligaments Very little anesthesia is required In the rare case where the aorta is opened spinal anesthesia is used

**Anesthesia in Operations for Neurovascular Lesions in the Upper Extremity** —Where operations are performed low in the neck the possibility of injury to the pleura always exists Endotracheal anesthesia therefore is the safest choice The possibility of hemorrhage requiring further operation or entrance to the thorax for control is another reason for the use of the closed tube type of anesthesia Cyclopropane anesthesia is the preferred agent.

**Anesthesia for Interruption of the Sympathetic System.**—*Lumbar Sympathectomy* —Spinal anesthesia is preferable to other types of anesthesia The negative abdominal pressure muscular relaxation and the ease with

which the peritoneum and its contents can be displaced with this modality is sufficient reason for its use The anesthesia level must extend to the sixth dorsal nerves Once this level is obtained the patient is positioned Hypobaric anesthesia permits an early positioning. The operative side is elevated by placing a sand bag under the hips. The arm on the operator's side is swung across and above the patient's body, thus pulling the lower ribs away from the crest of the ilium The table is then broken, increasing the spread in this area The position is secured by keeping the leg on the operative side straight and flexing the under leg A pillow is placed between the two legs to protect them A strip of 3-inch adhesive is run from one side of the operating table to the other crossing the greater trochanter on the operative side and the flexed knee on the other side This position permits the viscera to fall away from the operator The table break and this position brings the lumbar vertebrae close beneath the incision point. This latter centers at the second lumbar vertebrae level (2 inches above the crest of the ilium) The only technical difficulties that have been encountered in this procedure occurred when the spinal anesthesia failed or had to be supplemented by a general one Spinal anesthesia can be made isotonic with spinal fluid, or heavier or lighter than the solution by the addition of distilled water, dextrose, or one of the alcohol solutions When the solution is lighter, it rises in the spinal column dependent upon the position of the patient This permits an early positioning and elevation of the lumbar area Where the anesthetic is made heavy, one must gain the correct level by tilting the table up or down To make the solution *hyperbaric* (heavier than spinal fluid), 10 percent dextrose is added to the anesthetic agent For *hypobaric* (lighter than spinal fluid), the procaine crystals are dissolved in a triple distilled water solution Where longer anesthetic is required, a ½ per cent solution of pontocaine hydrochloride is used This solution is effective but more toxic than procaine Various combinations of procaine, pontocaine and the other derivatives of the anesthetic agents have been used and are detailed in books on modern anesthesiology [21]

*Thoracic Sympathectomy*—A closed endotracheal anesthesia is used whether the posterior or anterior approach is elected The patient is in a modified cerebellar position for the posterior approach to the ganglia

*Thoracolumbar Sympathectomy*—An endotracheal type anesthesia is essential inasmuch as the thorax will be opened

*Cervical Sympathectomy*—In the occasional cervical denervation of the sympathetic system, local anesthesia by infiltration and block has satisfactory There is no contraindication to a general anesthesia

**Anesthesia in Operations on Venous Lesions (Varicose Veins Thrombosis and Ulcers)**—In varicose vein operations and ope venous clotting or ulcers, spinal anesthesia is selected In most procaine crystals (1 per cent) permit an hour's anesthesia. In [illegible] sional prolonged operation pontocaine hydrochloride can be used [illegible] caine (3 to 5 per cent) is somewhat more toxic The anesthesi[illegible] continued for three hours For the obese, the introduction of th[illegible] with the patient sitting on the operating table with his feet at th[illegible] level of his hips facilitates the injection (Fig 5). Local anesthesia

used in vein ligations but does not permit painless vein stripping Since stripping between the incompetent points is now routine in most clinics the local infiltration method rarely is employed

The length of anesthesia can be increased using normal procaine by injecting the solution slowly so-called dribbling keeping the patient on one side and varying the level of the table With this technic we have been able to operate from three to four hours on 100 mg of procaine dissolved in spinal fluid without the addition of other drugs This concentrates the anesthetic solution at the level to anesthetize that one extremity alone.

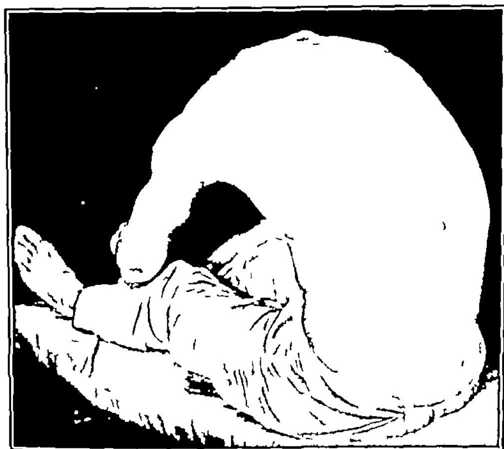


FIG 5 —Position for administering spinal anesthesia in heavy or obese individual Sitting position with legs on table at right angle to the trunk

Note how this position presents the spine processes even in a heavy individual

**Controlled Hypotension Anesthesia in Cardiovascular Patients** —If the tension in the blood vessels is decreased during the operation the blood loss likewise may be reduced There are advantages to such a technic as a more bloodless operative field is obtained Some disadvantages and dangers may develop When a hypotensive anesthesia is used the dangers are accepted Thus a hypotensive anesthesia is selected only when the danger from blood loss at a normal blood pressure is greater than the danger attending the hypotension Some operations previously impossible to perform have been completed successfully during a hypotensive state An example are some of the arteriovenous fistulas or aneu

rysms, particularly in the brain The use of hypotensive anesthesia, therefore, in essence becomes the selection of patients with an indication for its use

Efforts of both the surgeon and the anesthesiologist have been to keep the blood pressure, pulse and respiration as near normal as possible during the operative time The physiologic drop in blood pressure with such agents as spinal anesthesia was not accepted for many years Efforts were made to artificially elevate the pressure when it fell due to the sympathectomy effect of the anesthesia Adrenalin, ephedrine, neosynephrine, and other drugs in this category, were injected prophylactically and therapeutically through the years to counteract pressure drop One of the first to indicate that such a drop was not abnormal was Babcock[4], who pointed out that there was sympathetic denervation of a large part of the body during such anesthesia, with pooling of the circulating media Forceful efforts to make this blood circulate were abnormal and were resisted by the body as shown by the pounding of the heart, etc

The introduction of certain drugs which promote a physiologic hypotension was popularized in Europe and particularly in Great Britain. Their reported experience exceeded that of all other anesthesiologists. Of those replying to a questionnaire[39] sent to over 600 anesthesiologists, 59 per cent had used hypotension Only 4 per cent had abandoned it Over 21,000 experiences were recorded

TYPES OF HYPOTENSION—Hypotensive anesthesia may be obtained by an extended spinal anesthesia, the use of methonium compounds, refrigeration of the patient, deep ether or pentothal with postural changes, extradural blocks, and other methods such as the injection of sedatives or other anesthetic agents.

(*a*) *Spinal Anesthesia* —Spinal anesthesia may cause hypotension by raising the anesthesia level and therefore increasing the sympathectomy effect. With the modern drugs and technic, this can be done safely It is utilized in our Clinic most often by the continuous spinal anesthetic method In over 21,000 cases of controlled hypotension reported from Britain, this method was used in one-third of the times [39] The technic of its administration is known to all trained anesthesiologists but should be used only when an experienced, capable doctor is available to supervise it and counteract ill effects The complications of this method are those of all hypotension measures and in qualified hands should be no greater than with any of the hypotensive drugs

(*b*) *Methonium Compounds* —These drugs, hexamethonium and pentamethonium, drop both the systolic and diastolic pressure We have used them safely in both adults and children Their use is not advocated in patients who have hypertension, hypotension or advanced cardiac, renal or hepatic disease The methonium compounds were utilized in over half of the 21,000 cases reported in Britain

(*c*) *Hypothermia* —The early work of Fay[32] demonstrated the ability of the body to withstand a subnormal temperature Fay's patients were ones with hopeless carcinoma In dropping their temperature, he reduced their metabolism markedly Pain decreased and by this method apparently the growth of the tumors was retarded. Allen[1,2] and Crossman.

showed the toleration of the body for cold. Our own experiences confirmed the anesthetic effect of the cold inasmuch as legs could be amputated painlessly under this agent alone.[6-76] The general metabolism is lowered, the demand of the body for blood supply is reduced and a part sometimes can survive with cold applications better than when it is warm.

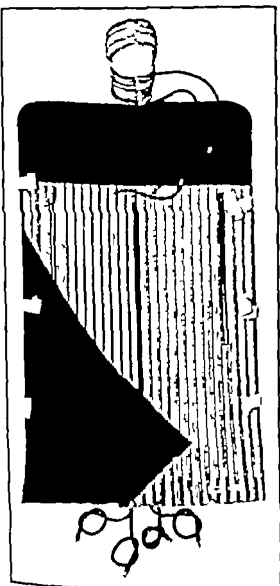
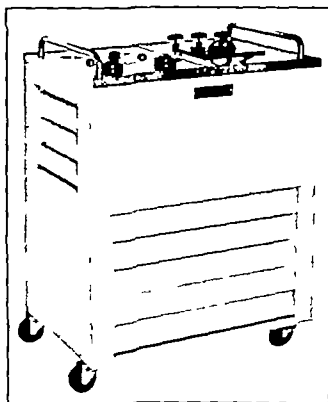


Fig. 6 – Rubber blanket for inducing hypothermia. Alcohol-water solution is pumped through rubber tubes by electric motor. Temperature can be regulated and reduced to 78° F. Patient can be warmed by a reversed method. (Manufactured by Therm-o-Rite Products Corp., Buffalo, N. Y.)



These efforts as well as the animal studies by Lang,[73 74 75] Schumacker, Lewis,[47] Radisch,[66] Talbott,[79] Cookson,[22] Neptune,[62] Bailey,[24] and Bigelow[11] led to an enlargement of the hypothermic program in efforts to reduce blood loss during operation and to counteract the reduced oxygen supply in cyanotic patients and those in whom the oxygen supply to the brain had to be reduced or shut off. McQuiston in Pott's[66] Clinic routinely cools cyanotic children undergoing cardiac surgery and reduces the amount of anesthetic agent necessary. Bailey[24] has reported similar results. The super

ior and inferior vena cava have been shut off for fifteen minutes in dogs in whom the temperature had been reduced to 24° C (76° F). We have used similar methods to produce hypotension and reduce the blood loss in operating on aneurysms and in other intrathoracic interventions Using the refrigeration blanket* technic we drop the temperature to 86° and 88° F This technic has worked well but there are certain complications which range from delayed reaction to pulmonary and cerebral complications The complications are similar to those following hypotension induced by other methods, and the indications and contraindications of this type of hypotensive procedure are the same as for the others The fact that the chamber of the heart may be made bloodless for a sufficient time to permit an intracardiac attack on a serious problem makes this type of procedure potentially important ..

(*d*) *Other Methods* —Some degree of hypotension has been produced by arteriotomy, extradural blocks, various local and sympathetic injections, venous sections and other sedative and anesthetic agents These measures do not appear as satisfactory for primary hypotension as those detailed The addition of such drugs as intravenous pentothal, curare and other sedatives as an adjunct to some of the other measures is valuable We have used it with controlled spinal anesthesia and with refrigeration, where it is valuable in eliminating the shivering which causes exhaustion and death Other barbiturates may be found to be equally valuable

LEVEL OF HYPOTENSION —Adequate hypotensive anesthesia can be obtained with a systolic pressure of 60 to 77 mm Hg Three-fourths of the 21,000[39] patients reported from Great Britain with controlled hypotension were kept between 60 to 80 mm Hg Posture is used as an adjunct to obtain the bloodless field To restore the normal blood pressure, some vasopressor agent is used routinely

COMPLICATIONS OF HYPOTENSION ANESTHESIA —Some complication[39] occurs in 3 to 4 per cent of the patients treated with hypotensive anesthesia The most common complication is delayed postoperative recovery Of the complications this one has been observed in 30 per cent The second most common complication was a reactionary hemorrhage which was observed in 29 per cent of the abnormal responses Difficulty with vision, anuria or oliguria, has been reported

| | |
|---|---|
| TOTAL NUMBER OF PATIENTS | 21,000 |
| TOTAL NUMBER OF COMPLICATIONS | 549 |
| The serious complications were as follows | |
| | *Approximate percentages*[39] |
| Delayed Postoperative Recovery | 32 |
| Reactionary hemorrhage | 27 |
| Difficulty with vision | 21 |
| Anuria or Oliguria | 14 |
| Cerebral Thrombosis | 3+ |
| Coronary Thrombosis | 2+ |
| Cardiac Arrest / Cardiovascular Collapse | Less than 2 |
| Other Complications were Mild | — |
| TOTAL | 100 |

* Therm-o-Rite Products Corp, Buffalo, N Y.

It is significant that 60 of the 84 renal complications occurred in those with a systolic pressure of 60 or below and that two-thirds of the cardiovascular complications were in the same pressure level.

MORTALITY OF HYPOTENSION ANESTHESIA.—There were 46 deaths in the 21,125 patients. This is a ratio of 1 to 459, or less than two-tenths of 1 per cent. The most common cause of death was cerebral thrombosis. Adding the coronary thrombosis and cardiac arrest, 14 deaths, or roughly 30 per cent, were in that category.

CONTRAINDICATIONS TO USE OF HYPOTENSION ANESTHESIA.—The contraindications to this anesthetic agent are obvious. There must be a specific and urgent reason for employing this measure which outweighs the disadvantages and dangers.

The contraindications are:

1. No indications *per se* for the hypotension anesthesia.
2. Patients in such obviously poor status that any drop in pressure may be fatal.
3. Inexperienced anesthesiologist or surgeon, or anesthetic difficulties.
4. Pregnancy, toxicity or shock.
5. The hypertensive or hypotensive patient.
6. An inadequate blood volume or inadequate blood replacement facilities.
7. Difficulty *with oxygenation or keeping airway patent.*

## ANESTHESIA IN CARDIAC SURGERY

I. **Congenital Heart Lesions.**—(*a*) PREMEDICATION.—The surgeon and the anesthesiologist have a double problem. These patients have hypoxia even at rest. Excitement, fear or crying may increase anoxemia to the point of unconsciousness or convulsion. Any increased demand for oxygen cannot be met due to the low reserve. In addition, all of these children *present a marked psychic factor due to their disease. It has been found* by Taussig and others[60] that infants tolerate morphine well, better than they do the excitement of an anesthesia. At two or three months an infant of 7 or 9 pounds can be given 1/16 grain of morphine and 1/300 grain of atropine. At four years a child can take 1/8 grain of morphine and 1/150 grain of atropine. Sedation thereafter is determined by age and not weight. From five years of age to puberty 1/4 grain of morphine and 1/100 grain of scopolamine is tolerated.[60]

Certain aids to the handling of such patients now exist. Besides the vital signs, continuous kymography (arterial pressure and pulse), continuous electrocardiography, oxymetry, and oxyhemographic readings, and blood volume, in addition to a computation of actual blood loss by weight, are available. Hyperthermia is a serious complication. The use of a continuous rectal thermometer is important. Reduction of the body temperature not only is safer for the patient but reduces the amount of anesthetic agent necessary. The infusion drip is begun after the anesthesia is started. This reduces the pain and fear factor. The circulation system must not be overloaded. The infusion is only a safeguard should hemorrhage occur.

Ether anesthesia remains the time-proven safe agent in questionable cases in children This is controlled effectively by intubation

The anesthesia problem in operations for congenital heart disease depends upon the presence or absence of cyanosis It is similar for the patients with patent ductus, coarctation of the aorta, anomalies of the aortic arch, or other congenital defects Where cyanosis is not present, there is a greater safety factor

The type of drug to be administered and the anesthetic agent for the congenital patient have been detailed under each such lesion To some extent the anesthetic choice depends upon the anesthesiologist's experience For cyanotic patients, cyclopropane is preferred because of the higher percentage of oxygen which can be administered The tendency towards arrhythmia is counteracted by intravenous procaine Where some contraindications or antithesis to this gas exists, ether is a time-tested, safe anesthesia for children in general Our objection to its routine use is its irritating action to the mucous membranes and the choking sensation experienced by the patient, with subsequent excitement and struggling which may have untoward effects on the patients whose cardiorespiratory system is inadequate Cyclopropane is preferred by the anesthesiologists working in the clinics where most of the cyanotic babies are operated In the non-cyanotic patients, this choice again varies with the individual's experience The emphasis should be on the use of the minimal amount of anesthetic agent which abolishes pain, permits an easy induction and a quick recovery. The avoidance of overdose of drugs is re-emphasized

**Anesthesia in Acquired Heart Lesions.**—This new and interesting type of cardiac surgery will be discussed in Chapter 10 The problem of anesthesia is important and may determine the success or failure of the procedure Again, there is a double problem of the patient with a poor cardiac reserve, at times in partial pulmonary failure, who is made worse by exertion, anxiety or fear Considerable experience has been accumulated in various clinics in the management of this problem

A *Preoperative Preparation*—Psychotherapy is extremely important The patient must be made a confidante, the operative success and his part and its importance in the ultimate outcome should be explained He should learn what cooperation includes He should have become acquainted with his anesthesiologist Where possible, a patient who has had a similar procedure with a successful outcome should talk to the patient The patient should be in the hospital a sufficient time to be familiar with the routine The control of the family is most important. The problems encountered in blood procurement, nursing care, etc, should be arranged but removed from the patient's mind entirely

(1) *Premedication*—It is better to use more premedication than to have the patient apprehensive Short-acting barbiturates and Demerol or morphine are used, the dose of each being individualized Collins[21, 23] favors sodium amytal, and follows this with morphine Ruth[23] uses nembutal or seconal and then Demerol with atropine These variations depend upon the anesthesiologist's experience The induction should be in a quiet atmosphere In this respect music has a place Armstrong, in "The Art of Preserving Health," stated, "Music exalts each joy, allays each

grief expels diseases softens each pain' I can recall the white gramaphone in the operating room of my late uncle Dr Robert Emmett Farr[20,21] All of his surgery was performed under local anesthesia and his "psycho-anesthetist the young lady who sat with the patient chose the music in accord with the patient s desires and moods Collins for years has added his own vocalizing during induction[21,22]

(2) *Infusion* — An infusion into a brachial anterior tibial or saphenous vein is necessary This is placed and kept open for use only in event of hemorrhage Again the surgeon and anesthesiologist must be cautioned against overloading the circulation In an acquired heart lesion the margin between the competence and incompetence can be lost readily by increasing the circulating volume even slightly

(3) *Induction* —Surgeons and anesthesiologists vary in the technic of induction We favor a cyclopropane induction as the quickest and least psychically shocking This is aided by the 0 1 per cent procaine intravenous solution and at times a small amount of pentothal

(4) *Intravenous Procaine* — Intravenous procaine has definite antiarrhythmic effects[12] These appear more constant and controllable when the drug is given by vein than when it is applied topically on the heart The side effects of this drug are stimulation of the central nervous system and certain anaphylactic effects on the circulation of a deleterious nature Of the some 60 analogs of procaine procaine amide (known as pronestyl) has the most prolonged effect It is slowly metabolized and excreted by the kidneys for the most part in the same form Intravenous use of procaine may prevent ventricular fibrillation reduce ventricular tachycardia and minimize the reactions to the intracardiac manipulations necessary in cardiac surgery It is important to realize that hypoxia itself can produce arrhythmia *No drug can counteract the lack of oxygen* and its supply should be maintained adequately

B *Anesthesia* —Many anesthetic agents have been used Bailey[4] and Glover[24] resort to fifty fifty nitrous oxide (50 per cent nitrous oxide—50 per cent oxygen) to supplement their induction with pentothal and the anesthetic actions supplied by procaine Curare is added at times

After considerable experience and discussion our Anesthesiology Department uses a mixture in which 1 liter of nitrous oxide is combined with 1 liter of oxygen and 500 cc of cyclopropane for induction With this combination and the intravenous procaine the myocardial reaction is slowed and arrhythmias are prevented It appears significant that the two clinics where most of the operations on cyanotic patients are performed utilize cyclopropane[16,17]

The anesthesia is maintained by endotracheal cyclopropane The objective is to use the minimal amount of the drug which allays the patient s apprehension removes pain and permits the patient to recover as soon as possible after the surgical operation is completed The basic principle is to administer an anesthesia through an endotracheal tube by a to-and fro absorption technic with controlled respiration The purpose is to assure an adequate airway a high level of oxygen in the trachea and bronchi, and a low level of carbon dioxide This makes use of the partial pressure gradient[23,24]

The poison curare, for many years dabbed on the arrow tips of the South American Indian to paralyze his foe, has had a therapeutic place in medicine and particularly in anesthesiology Muscle relaxation follows its therapeutic use The dangers of apnea, however, are definite and death has followed its injection It cannot be advocated, therefore, as a safe adjunct in patients whose underlying disease may predispose them to apnea

Curare[34,35] should be used sparingly if at all in this group of patients. The anesthesia should be lightened as the chest is closed The lungs should be inflated Positive pressure aeration must be continued until the patient is breathing freely spontaneously. All secretions should be aspirated using a catheter and suction The bronchoscope should not be employed unless the catheter fails An airway should be maintained as long as necessary The patient is placed in an oxygen tent and encouraged to cough and move about Subsequent care has been detailed under treatment of acquired cardiac lesions, pages 58 to 62

See pages 54-56 for Bibliography

Chapter

4

# BLOOD AND FLUID BALANCE

THE necessity of replacement of fluids lost before and during operation due to sweating, starvation, urinary and rectal loss is well known to all surgeons and anesthesiologists. This problem has been worked out to a point where so many cubic centimeters are returned for every hour of operative intervention and degree of temperature rise. It has also been standardized for the patient's weight, age and status of his cardiovascular and respiratory and renal tracts. This subject needs no further exposition at this time.

Blood replacement cc. for cc. and liter for liter is a newer concept of fluid control. Recognition of this necessity has placed many new operations in our potential surgical field and reduced the dangers and complications in others. The body appears able to compensate for the loss of plasma, but it is limited in its ability to restore red cells. Not only is there blood loss in the operative field, but there is a depletion into the tissues, into the surgical specimen, and into vessels proximal to ligatures, these ligatures immobilizing blood and thus losing it to the circulating volume.

An insidious amount may be lost postoperatively. As a result of this blood loss a hypoxia occurs because of loss of oxygen-carrying red blood cells. The vital signs often do not show this loss until such time as depreciation is cumulative to the shock stage. Shock at such stages may be irreversible even by replacement. The common practice, therefore, of beginning fluid and blood transfusions when the pulse rises or the blood pressure drops is clinically unsound in the patient with a normal circulatory system and may be physiologically fatal. A personal element enters this picture. The surgeon may feel that the need for blood transfusions is a reflection on his technic. He often will minimize the blood loss or even deny obvious hemorrhage. Subsequent shock, no matter what its cause, may be blamed on the anesthesiologist. This blood loss problem may be stated:

1. Blood loss always is greater than anticipated or estimated.
2. Procrastination in replacing blood loss never is to the advantage of the patient.

The blood loss should be estimated by weighing sponges and towels against their dried weight, compiling loss in clots, and measuring aspirating bottles. A gravimetric replacement method should be instituted and this should be at least the minimal amount replaced. This measure does not estimate the loss into the tissues or into the ligated vessels.

It has been estimated that there is an average 1000 to 1500 cc. blood loss in entering the chest.[14] This figure seems conservative. Osmetric and

blood volume determination will make such estimates more accurate, but clinical evaluation still will be important.

Blood replacement must be tempered by common sense For example, the patient with an arteriovenous connection for any length of time will have developed an increased circulating media volume Closing such a fistula will throw an extra load suddenly on the heart Heart failure has been the result In such patients the blood loss is helpful and even phlebotomy may be necessary In a like manner, patients with a diseased heart stand mild blood loss better than they do an overload It has been demonstrated that patients with diffuse coronary arteriosclerosis cannot withstand the increase in the blood volume attendant to the administration of several liters of blood [46] The aged, the diseased, those with a poor myocardial reserve, and the patient with pulmonary râles or edema, thus evidencing pulmonary failure, must be treated in a special and selected manner The same problem exists in those with renal disease where normal elimination is impossible or delayed [14] These patients must not have their circulatory systems overloaded

**Intra-arterial Transfusion.**—Intra-arterial transfusions may be a life-saving procedure In acute hemorrhage the blood volume loss and the attendant shock reduces the amount of the circulating media reaching the heart for redistribution to such an amount that the heart has insufficient volume to transport and against which to contract The intra-arterial transfusion helps correct this problem A volume of blood is transfused through the artery toward the heart Ventricular systole then can send this blood component distally and the contraction of the cardiac muscle can be kept forceful and regular The volume necessary for continuing the heart's action is much less The radial artery is the preferred blood vessel for such arterial transfusions, but in an emergency any available artery, including those in the operative field, can be utilized The author has used the aorta and, on occasion, the left ventricle of the heart

The principle of the intra-arterial transfusion itself is simple A pressure greater than the systolic blood pressure is necessary to introduce the blood This can be supplied by an apparatus attached to a blood pressure cuff Due care must be exerted to have an adequate fluid reserve or protecting container between the pressure of the cuff and the artery so that air does not suddenly enter the arterial tree and become an embolus Several such apparatuses have been devised for intra-arterial transfusions One described by Ziccardi and Madden[56] has worked well More simple ones also are available and these can be constructed easily See Fig 7

*Technic of Arterial Transfusion* —The artery to be used is selected. In most instances the radial artery is selected due to convenience and the fact that the ulnar and not the radial is the end artery in the hand Where this artery is not available, another is selected The artery is exposed under local infiltration anesthesia with 1 per cent procaine, this latter anesthesia aiding in the reduction of arterial spasm A suture above and below the injection site will elevate and tense the artery for the puncture The adventitia may be cut The #15 or #16 gauge needle is introduced parallel to the artery into the artery A Landemann stylet or regular beveled needle may be used The closed pressure system is attached This may be esta-

blished with a blood pressure apparatus distal to the closed blood bottle To introduce the blood the pressure must be greater than the patient's mean blood pressure In case of circulatory arrest the pressure may be raised to 50 to 70 mm of mercury This will cause a rapid infusion If only slight need exists for the blood flow the pressure may be as low as 10 mm of mercury above the patient's mean blood pressure Where only a pressure cuff is used to supply the pressure gradient one must guard carefully against the exhaustion of the blood flow and the introduction of air A middle container of blood or saline can be placed as a safeguard

Some arterial transfusion systems are conducted with the pressure at approximately mean pressure (approximately 100 mm Hg) When the pressure of the patient falls as with hemorrhage then the mean pressure automatically injects the arterial blood Such a system requires close observation to prevent air entrance

**Autotransfusion.**—Highmore[41] is credited with the suggestion for an autotransfusion Duncan[29] probably performed the first one By 1936 275 autotransfusions had been done in America Since the mortality from hemorrhage is proportional to blood loss autotransfusion has many possibilities Watson[83] reported only 1.5 per cent mortality Brenizer[18] detailed a method of collection of blood loss for retransfusion Such a procedure is recommended where there is a continued blood loss It has been tried in the hemorrhage accompanying the wiring of aortic aneurysms.

**The Blood Supply Problem.**—The mass use of whole blood for transfusion and the incorporation of the procedure into operations previously performed without transfusion has made the problem of blood supply in blood banks an acute one In addition the routine use of this modality during World War II and the Korean episode depleted the donor sources of many blood banks It has been shown that red blood cell suspensions can be given instead of transfusions This saves the plasma for other uses and also reduces the overload reaction in cardiac and pediatric patients In addition where needed the patient can receive a transfusion of red blood cells and a later one of plasma This technic is indicated for the iron deficiency anemias the anemia where an increase in blood volume is contraindicated (cardiac kidney or hypertensive patients) and the anemia where oxygen carriers alone are needed

**Plasma Expanders** —*Plasma expanders* have an important place in the replacement of whole blood deficiency [77] These fluids with a gel basis can be produced at the rate of two million pints per month The impossibility of supplying the blood needed in event of a major disaster such as atomic warfare has been recognized by both civilian and medical authorities With the minimal drain relatively placed on our blood supply by the events in Korea we see that blood substitutes must be obtained Hogan[44] reported on the use of gelatin in shock in 1915 Little interest followed until World War II when the need arose acutely The substances have been purified and are now available for safe use Gelatin for intravenous injection is prepared and refined from beef bone It is pyrogen free and partially hydrolyzed by steam autoclave for twenty minutes at 15 pounds pressure [43] The substance is a gel at room temperature but melts at 85° F A 6 per cent solution in physiological salt solution is used Due to its shape there

is little tendency for the molecules to leak out of the blood stream It reaches its height in increased volume in four hours after injection [43] It thus draws extravascular fluid into the blood stream Cardiac output returns to normal after twenty-four hours at which time 50 per cent of the gelatin is still in the blood stream It disappears in forty-eight to seventy-two hours It is easily stored, inexpensive and nontoxic It should *not* be used where decompensation is feared for obvious overload reasons Its use has been clinically successful [43] It has been used intra-arterially

*Other Plasma Expanders* —Dextran, a polysaccharide of dextrose, and peristran, a polyvinyl plastic, are being investigated as plasma expanders Dextran, a macromolecular substance, has had clinical use and is preferred in England [15a] Its usefulness is accepted

**Dangers of Blood Transfusion.**—With the commonplace utilization of whole blood transfusion in modern surgery, the fact that there are certain dangers attendant to its use often is forgotten Where it is lifesaving, of course, there should be no question of its use Where the operation is not shocking, or where there is not a severe loss, the possible complications must be remembered

*Anuria* —Anuria is a serious complication Murray [61] reported 100 such complications in a year The transfusion should be terminated if there is any reaction A limited intake of fluid is a primary requisite in the treatment The electrolyte balance must be maintained, and alkalization helps in the therapy The $CO_2$ combining power may rise to 80 The urethral catheter lavage of the renal pelvis helps in the first twenty-four hours A high carbohydrate and low protein diet should be added In addition, oxygen therapy, the antibiotic drugs, and vitamins, particularly the B complex and C, help counteract the effects For the muscle spasm and tetany which may occur, calcium gluconate is helpful If the condition progresses despite such therapy, decapsulation of one kidney is advocated [28] (See p 54-56 for bibliography )

Chapter

# 5

## CARDIAC ARREST AND RESUSCITATION

MAN may die in stages We have all seen the death of a limb of a part or most of the brain, the cessation of kidney action and the obstruction of the intestinal tract The cessation of the cardiac action however is the final step Circulatory obstruction follows with cessation of blood supply to the vital centers The brain cannot stand anoxemia The brain shows irreversible changes if it is deprived of oxygen for more than three and a half minutes and permanent changes after seven minutes[41] It seems certain that a defeatist s attitude toward stoppage of the heart s beat probably has resulted in the loss of many patients It is understandable that when the heart sounds are no longer heard in an advanced carcinomatous patient or an elderly cardiac death is expected accepted and respected A sudden cessation of heart beat however in the patient who does not have other cause for sudden death should be a challenge and demand for active aggressive action in an attempt to restore the cardiac impulse. Our reaction must be as natural and as prompt as when we tap an electric light bulb which suddenly turns dark in an attempt to restore the continuity of the tungsten wire and revive the light This action is similar to the mild shaking of a clock when it suddenly ceases to tick. We have learned how prompt must be our response to a failed or faltering respiration in the newborn The reaction of these infants to even mild stimulation is usual unless that intervention is delayed The operating room and the surgeon must be as attuned to emergency action when the heart ceases as they are to controlling serious hemorrhage

One year after anesthesia had been introduced the first death was reported A review of the findings[11] suggests it was cardiac arrest One hundred forty three patients with cardiac arrest had been reported up to 1945[7] The relative increase in the number of patients recently probably is due to our earlier diagnosis of this condition The expectancy of death during an operation with any type of anesthesia is less than one in a thousand Cardiac arrest makes up a high percentage of such patients This small group however are nearly always fatal unless the diagnosis is made immediately the correct therapy instituted within the minute and continued until the definitive result is certain The few patients who are restored by delayed action usually live with the mental deterioration coincidental with the anoxemia the so-called vegetable life

**Etiology of Cardiac Arrest.**—(1) *Overdose of Anesthesia* —Too much anesthesia is one of the most common causes for cardiac arrest If a patient with a normal heart receives more anesthetic than it can tolerate,

cardiac arrest follows. The problem in such instances is to produce an artificial circulation and respiration until such time as the anesthesia is removed or destroyed sufficiently to permit normal action of the heart. All of these patients should survive a cardiac arrest if the diagnosis is made early and the therapy is immediate, complete and continued.

(2) *Sensitivity to Anesthesia.*—The patient may be sensitive to a general or local anesthesia in certain concentrations and respond to it like those patients described under "overdosage" of anesthesia.[11] This may occur with small amounts of the anesthesia.

(3) *Anoxemia.*—It is known that anoxemia may sensitize the heart and cause an increase in the circulating epinephrine. Such lack of oxygen causes the carotid sinus to inhibit the normal cardiac activities.[27]

(4) Reflex phenomena (of the vago-vagal type)[48] at times causes the heart to stop. Stimulation of the vagus nerve inhibits cardiac activity.[69]

(5) Intravenous barbiturates and cyclopropane gas in high dosages[18] have a parasympathetic effect on the heart and may cause the heart to stop.

(6) Certain anesthetics such as cyclopropane, chloroform and ethyl chloride sensitize the heart to circulating epinephrine or other arteriospastic stimulants.[70]

(7) *Spinal Anesthesia.*—This agent may cause a cardiac arrest by sensitivity of the patient to the drug. The upper extension of the drug may have an indirect effect and cause cardiac arrest by inhibiting breathing due to paralyzing the thoracic nerves. The diaphragm contractions will also stop when the anesthetic level reaches the fourth cervical roots. The second, third, fourth and fifth thoracic sympathetic ganglia are supposed to contain the cardiac accelerator functions. Their bilateral paralysis may cause cardiac arrest. This latter fact has not been proven inasmuch as bilateral sympathectomy has been performed on these fibers without heart stoppage.

*The most common cause for cardiac arrest is hypoxia.* Overdosage of anesthesia or an inadequate airway are collaborators in this alarming catastrophe.

The incidence of this condition is difficult to determine. There were only 7 incidences reported at the Lahey Clinic in thirteen years.[51] A great deal depends upon the accuracy of the diagnosis, and the figures vary from Bailey's 2 each year,[6] to Cooley's[26] 48 in 878 operations for pulmonary stenosis. The incidence is greatest in intrathoracic operations in general,[26, 71, 55, 57] and in particular in operations on the heart and great vessels.

Many other causes for cardiac arrest have been listed. It is obvious that age, myocardial damage, extensive and traumatic surgery, hemorrhage, the action of the barbiturates by a parasympathetic action, pyrogenic and transfusion reactions, as well as any variation in the chemical content of the blood, may contribute to the picture.

**Pathogenesis of Cardiac Arrest.**—The heart responds to stimuli by rhythmic changes. These are determined by the myocardium, the oxygen supply and the stability of the neuro-regulatory system. The most common rhythmic change is tachycardia. Bradycardia and the various arrhythmias may result in cessation. This cessation may be by cardiac

dilation and stop in diastole ventricular fibrillation auricular fibrillation with ventricular arrhythmias or cardiac cessation in systole Of these the most common is standstill in systole [illegible] Ventricular fibrillation has the poorest prognosis but Beck Johnson and Lampson all have reported successful resuscitations [10,19,35] Many times the actual cause of cardiac arrest is not apparent The author has had one patient with a cardiac arrest during induction of spinal anesthesia without other known causes At times a combination of factors may exist

**Diagnosis of Cardiac Arrest.**—The surgeon often is not aware of the condition at once Should the operation be performed on the heart or a blood vessel however he may be immediately aware of cessation of the systolic thrust The condition may occur during induction as well as the maintenance of anesthesia It has occurred with all types of anesthetics in all age groups and under all circumstances of premedication The type of operation does not mitigate the likelihood of this complication

If the anesthesiologist has any suspicion he should call it to the surgeon s attention at once In a few patients there may be premonitory signs Bradycardia [illegible] tachycardia or other arrhythmias are precursors to cardiac arrest [17] A continuous electrocardiographic tracing is of great help The surgeon should make a confirmatory diagnosis in thirty to sixty seconds If there are any arrhythmias the gas should be washed out of the machine and oxygen administered Atropine may eliminate bradycardia Arrhythmias respond to 1 per cent procaine[illegible] or an amide of procaine [17,21,64] pronestyl and can be given in large doses intravenously [40]

The disappearance of the pulse and blood pressure are the primary symptoms These should be checked immediately by the surgeon An immediate decision as to intervention must be made and no procrastination is possible To paraphrase He who hesitates is lost if the surgeon hesitates the patient is lost

**Treatment of Cardiac Arrest.**—The material needed for opening the chest *MUST* be available One cannot wait for the procurement or preparation of the instruments A cardiac arrest set must be kept in a state of immediate readiness

In addition to the necessary instruments to open the chest the cardiac set should contain the following

(*a*) Syringes—4 (10 cc.) 4 (2 cc )
(*b*) Needles—4 spinal needles size 20, 10 (2 in long) size 20
(*c*) Ampoules—6 of adrenalin (0 1 per cent) 6 of calcium chloride (5 per cent) 10 cc. 6 of potassium chloride (5 per cent) 10 cc
(*d*) Flasks—pronestyl 1 per cent procaine solution
(*e*) Barium chloride solution freshly prepared 0.5 per cent
(*f*) An electric defibrillator or shock apparatus

Anderson [3] Harken [44] Beck [8] and Collins[22] all have made similar recommendations

In respect to the drugs the experimental work has shown that epinephrine hydrochloride is the best cardiac stimulant [8] Calcium chloride is the second best and is better than barium chloride or isopropylepinephrine.[42] Dogs placed in ventricular fibrillation by overdose of ether or chloroform could be defibrillated by a combination of cardiac massage

with electrical shock In only one of a series of instances was procaine needed as an adjunct The hearts were allowed to fibrillate for from three to sixty-two minutes while they were massaged. They were then electrically defibrillated and all recovered The most important step is the *cardiac massage* and defribillation The second most important is the injection of a cardiac stimulant

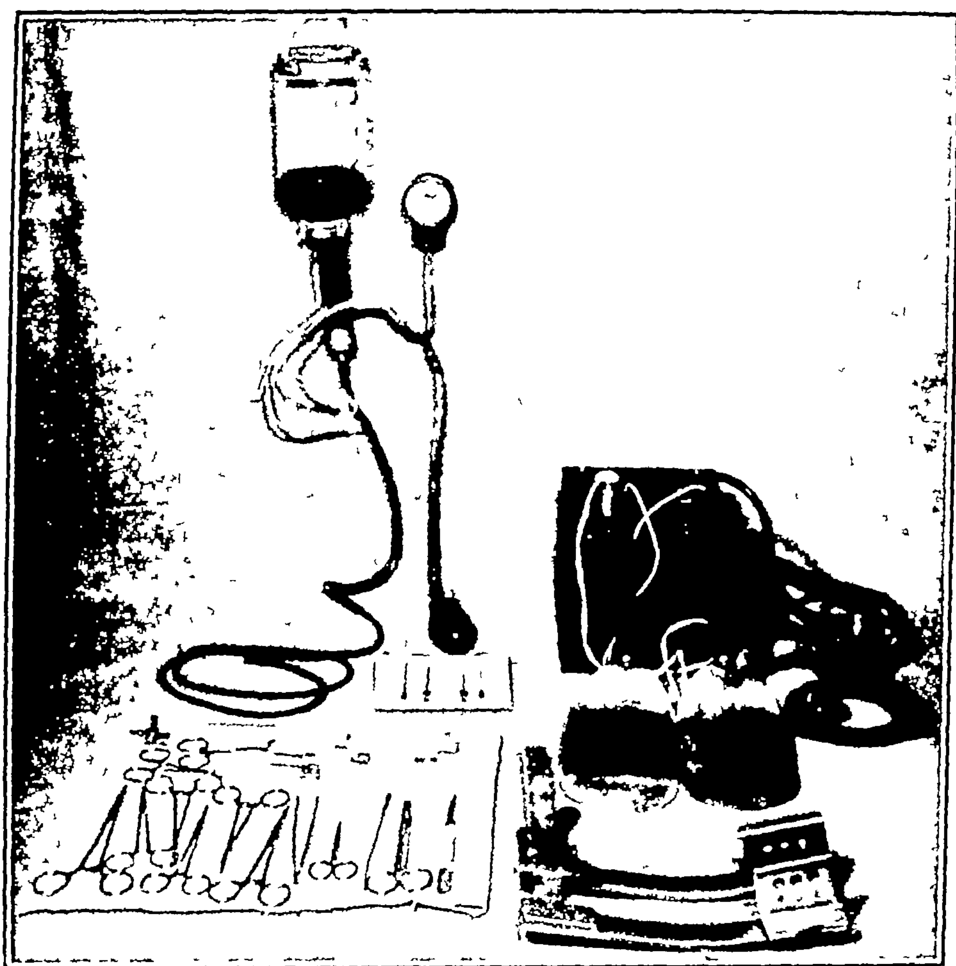


Fig. 7 —Instruments and apparatus for cardiac arrest Instruments on towel for cut down on artery Syringes and needles for injections Same instruments can be used for opening the chest Simple intra-arterial transfusion method using blood pressure cuff Rib spreader (Finnochietto) keeps the chest *open and protects* the hand during cardiac massage Electric defibrillator to bring the heart rhythm out of fibrillation

Prolonged massage may cause muscle injury or endocardial ecchymoses The surgeon's scrub nurse and team must be ready to act reflexly in such an emergency as three and one half minutes can pass too quickly in the confusion of an unprepared team

1. Cardiac puncture and aspiration may be performed at once if the syringe and needles are available To this can be added the injection of procaine and epinephrine Some patients acting like cardiac arrest are not actually in this state and will respond to this therapy. This step is indicated only if the syringes, needles and solutions are on hand (not in another room)

2 Artificial respiration should be performed and continued by positive pressure instituted by the anesthesiologist using 100 per cent oxygen

3 Cardiac massage This step is primary the most important and the only one which will resolve the process from inevitable death in nearly every case It is important to realize that artificial respiration alone is insufficient The cardiac massage must be instituted promptly and continued

**Technic of Treating Cardiac Arrest** —Time is not allowed for skin preparation although if a soap Hexacholorophene preparation is available this may be smeared on the surface The fourth or fifth left interspace can be opened at once The heart can be grasped and rhythmic cardiac massage begun without opening the pericardium It is compressed with a milking action from the apex to the base If the heart is too large it can be compressed against the sternum In some large hearts it may be necessary to use two hands. The rate of massage has been the subject of considerable discussion [8,38,44] Gunn suggested gradual compression and abrupt relaxation with never a greater rate than half that of normal [38] The essential point is to maintain the circulation and stimulate the heart The effort actually is to start the heart beat This develops from the massage and the use of epinephrine Epinephrine apparently never starts cardiac contractions although it can increase the strength of them if they are present [8] Procaine is given with the epinephrine

*Posture* —The head and chest should be depressed and the legs elevated This has the gravitational effect of improving the venous return without increasing cerebral congestion [37]

The possibility of an electric stimulator to act as a pacemaker has been studied by many [45] It seems likely that such an apparatus can be constructed Beck[10] has suggested the use of a resuscitation apparatus His efforts to have suction cups for cardiac massage proved no better than if as good as the manual method The time of continuing the cardiac massage is undetermined As long as there is hope that the action of the heart can be restored massage should be continued Touroff[56] had a successful restoration after forty minutes Leahy and Ruzicka[51] reported a survival rate of all 13 patients in cardiac arrest although 1 patient died the following day Only 5 or 38 per cent however had a normal mentality thereafter Bailey[47] revived 13 out of 40 of whom 4 survived In Barber and Madden s[7] reported group 48 out of 143 or 33 per cent, recovered completely

Beck recommended that the surgeon learn what to avoid [8] These are worthy of repetition He mentioned (1) do not listen for heart sounds (2) do not be afraid to open the chest because the heart may be vigorously pulsating (3) do not wait for an EKG (4) do not wait to inject epinephrine (5) do not dilate the rectal sphincter (6) do not try mechanical compression of the chest (7) do not give an intravenous or intra arterial blood transfusion. These steps will occupy so much time that the patient will be dead

Certain do s go along with the don ts These are

1 Introduce a proper fitting open tube into the trachea This should inflate and deflate the lungs with oxygen regularly by compression of the rubber bag

2. Open the chest and squeeze the heart. This completes the emergency. The fourth interspace can be opened at the left side of the sternum, the cartilage above and below divided if necessary, and the wound pulled open by the assistant. After the heart has been squeezed, and only then, should a self-retaining retractor be placed to give the surgeon wrist room.

3. In ventricular asystole, the heart may begin beating with massage alone. If it does not, epinephrine and procaine may be injected directly into the heart through the ventricle. The injection should consist of 5 to 10 cc. of a 1:10,000 solution of epinephrine and 5 cc. of a 1 per cent solution of procaine. These injections can be repeated if necessary. In ventricular fibrillation, the use of procaine and epinephrine together with the application of electric shock is advocated by Beck.[9] The heart has been defibrillated without the use of shock. The injection of the solution into the cavity of the right ventricle and massage alone may be sufficient.

Cardiac massage may be performed subdiaphragmatically when the patient is having an abdominal operation. This is somewhat difficult for the inexperienced surgeon. The diaphragm also can be divided. If there is any difficulty with either of these measures, the thorax should be opened promptly. It is considered helpful to occlude the thoracic aorta during the cardiac massage. This increases the coronary circulation and that to the brain. Carter's[20] recent experience in which there was a recovery after twenty-five minutes of cardiac arrest detailed the importance of manual occlusion of the aorta distal to the subclavian arteries. It was his feeling that this occlusion permitted sufficient oxygenation and circulation of the brain and coronaries to permit recovery.

**Prognosis in Cardiac Arrest.**—The fact that the heart can be started again is not new. Schiff[72] revived dogs by manual massage eleven and a half minutes after the heart had stopped from an overdose of chloroform. Prus[67] killed 100 dogs by electricity, suffocation or chloroform. Massage restored 14 per cent of the dogs whose hearts stopped due to electricity, and of the others, 75 per cent were resuscitated. His heart massage and artificial respiration was begun from fifty-five seconds to one hour after it ceased to beat. The first successful revival of a human patient was reported in 1902 by Starling and Lane.[75] Published figures from 1945 to 1950 show that after cardiac arrest 60 per cent[15] recovered, of which 30 per cent have a partial recovery. It is important to recognize that even with cardiac function re-established, recovery is not assured until respiration has returned to normal. Artificial respiration must be continued until this stage is achieved. The eventual outcome depends upon age, cardiac status, and the cause of the cardiac arrest. The most important factor, of course, is the rapidity with which the therapy was undertaken and the efficacy of the treatment.[19] The patient may recover his respiratory and cerebral functions completely. He may also be revived, but without cerebral adequacy. In certain cases, neither the respiratory nor cerebral functions returns. In the light of our present knowledge, these patients "live" only as long as their respiration and nutrition and elimination are maintained, and eventually must die of some intervening complication. A case in point is illustrative.

N. S., w. a. m., age twenty-seven, a lieutenant in the Air Force on leave before advanced training with jet planes, had sudden dizziness, headaches and unconsciousness. He was admitted to a service hospital where physical findings and spinal tap confirmed a diagnosis of cerebral hemorrhage likely due to a cerebral arteriovenous aneurysm. A second attack of unconsciousness was associated with a respiratory paralysis and cardiac arrest. With artificial respiration and intracardiac stimulation the heart regained its function. The unconsciousness persisted as did the respiratory paralysis. The patient was placed in a respirator after which his normal color returned. With cut-down intravenous feeding and continuous catheterization his electrolyte balance was maintained. Increased intracerebral pressure was controlled by atrophine and the insertion of a polythene catheter for drainage. The patient was maintained in this status for nine days. There was no return of consciousness and no effort at spontaneous respiration, but with the artificial respiration and intravenous feeding combined with the release of the intracranial pressure his vegetable-like existence was maintained. Whenever he was removed from the respirator cyanosis developed and he appeared dead. Upon reinsertion, his previous satisfactory status prevailed. It was a hopeless situation but demonstrated well the capacity of the functions necessary for life to be maintained separately. From such an experience one gains a respect for the heart and its ability to withstand great insult and the loss of all coordinating parts of the body on a spontaneous basis, such as the respiratory system, the brain and the alimentary and elimination channels. The possibility of operating on a patient who is in precarious physical by providing for a continuation of his respiration mechanically and a controlled electrolyte balance is presented by observing such a patient. (See pp. 54–56 for bibliography.)

Chapter
6

# ANTIBIOTIC THERAPY

THE advent of antibiotic therapy marked a milestone in medical and surgical progress No event in surgery has been more important since the introduction of anesthesia Operations previously inconceivable are now not only possible but elective We are only on the threshold of utilizing the potentialities of this discovery. Most likely, only the surface of available antibiotics has been uncovered Since the success or failure of so many cardiovascular diseases depends upon the control of infection, this part of the therapy is important.

1 *Active Sepsis.*—Where infection is present, as in occlusive arterial disease, thrombitis, or in surgical wounds, the organism should be cultured and its resistance to the available antibiotics determined in the first twelve to twenty-four hours The antibiotics to which it is susceptible should then be employed to a full therapeutic level In the meantime, the antibiotics which affect certain organisms should be started Exceptions exist and certain strains of organisms vary in their reaction to the antibiotic therapy in general and at certain times The importance of this therapy, however, cannot be overemphasized

2. *Prophylactic Therapy*—In the absence of a culturable organism but where inflammation or infection exists, *i e*, thrombophlebitis, the antibiotics may be given empirically. In like manner, where the operation to be performed predelicts the patient to some infection, *i.e*, cardiac surgery, the same indication for antibiotic therapy exists.

3 *Topical Application.*—Infected areas such as ulcers may respond to the local application of antibiotics.

4 *Contaminants, i e, Fungus, etc*—The trichophytins, epidermophytins, etc, are universally present A skin break, particularly in the lower extremity, permits their entrance At other times their irritation, or the scratching associated with them or other causes, permit a portal of entry for a secondary infection. Such contaminants should be treated by mild fungicides No effort should be made to "cure" such fungi by the use of salicylic or benzoic acid or strong *x*-ray therapy because of the deleterious effect of these agents upon the skin Strong antiseptics are contraindicated for the same reason

A list of the antibiotic agents most often utilized in the treatment of cardiovascular lesions follows The organisms which respond most often to these various drugs is appended The dosages mentioned are suggested ones for the adult These must be varied with age, physical status, blood components, and the status of the cardiovascular, renal and liver systems

KEY A Drug of first choice
B Effective
C Only moderately effective
D ? May prove to be agent of choice

| | *Penicillin* | *Dihydro-streptomycin or Strepto-mycin* | *Aureomycin or Terra-mycin* | *Chloram phenicol* | *Sulpha* |
|---|---|---|---|---|---|
| I *Gram Positive* | | | | | |
| 1 Hem. Strept | | | | | |
| Group A | A | | B | B | B |
| Group D | A | A | B | C | |
| Others | A | | B | B | |
| 2. *Streptococcus viridans* | A | | B | | |
| 3 Staphylococcus | A | B | A | B | B |
| 4 Pneumococcus | A | | B | B | A |
| 5. B anthracis | A | | | | A |
| 6. Clostridia | C (Plus Serum) | | | | |
| 7 C. diphtheriae | A (Plus Serum) | | B | B | |
| II *Gram Negative* | | | | | |
| 1 Meningococcus | B | | B | | A |
| 2. Gonococcus | A | B | B | B | B |
| 3. E. coli | | B | A | A | B |
| 4. A. aerogenes | | B | A | A | |
| 5 B proteus | | C | | | C |
| 6 Ps. pyocyaneus | | C | | | |
| 7 *Salmonella* | | | B | A | |
| 8 Shigella | | B | A | A | |
| 9 Brucella | | A | A | B | |
| 10 P tularensis | | A | B | B | |
| 11 H. influenzae | | B | D | D | |
| 12. H. pertussis | | | A | A | |
| 13. K. pneumoniae | | A | A | B | C |
| 14 Granuloma inguinale | | B | D | D | |
| III *Acid Fast* | | | | | |
| 1 M tuberculosis | | A (Plus PAS) | | | |
| IV *Spirochetes* | | | | | |
| 1 Syphilis | A | | B | B | |
| 2. Leptospira | C | C | D | | |
| 3 Spirillum minus | A | | | | |
| V *Viruses* | | | | | |
| 1 L. venereum | | | A | A | B |
| 2 Trachoma | | | A | C | B |
| VI *Rickettsiae* | | | | | |
| 1 Rickettsiae | | | A | A | |
| VII *Actinomycetes* | | | | | |
| 1 Actinomycetes | A | | | | |

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Under certain circumstances these drugs cause a leukopenia or anemia. An agranulocytosis has developed. In some cases there has been a restriction, a retardation, or even a complete shut-down in the blood-forming mechanism It is of importance, therefore, for the surgeon to check carefully and regularly on the blood status of his patient if he is under antibiotic therapy The possibility of making the patient "fast" to one of the drugs exists, and these drugs should not be used without ample justification

*Co-Use of Antibiotics and Antithrombotic Drugs*.—The antibiotic drugs sterilize the bowel The synthesis of vitamin K depends upon the action of the intestinal bacterial flora. Prolonged use of the antibiotic drugs may deprive the body of vitamin K and cause an increased bleeding tendency. This is of more importance if antithrombotic drugs are administered jointly with the antibiotic drugs.

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# SECTION III

## Surgery of the Heart

## Chapter 7

## CHEST DYNAMICS

*Cardiac Physiology, Opening and Closing of the Chest Postoperative Care Cardiac Findings in Disease*

**Introduction.**—There is need for defining what is meant by the term cardiac surgery " In the past operative attack upon the heart itself was confined to the surface or walls of the heart or the great vessels arising from it. This field has enlarged rapidly to include the operations on the coverings of the organ the pericardium the blood supply to the heart muscle and internally in the heart on the valves openings growths clots and congenital defects Cardiac surgery now includes

(1) *wounds of the heart*

(2) operations on the pericardium

(3) operations *to improve or substitute for the coronary blood flow* to the myocardium

(4) operations to by-pass a section of the heart by *anastomosis of the* inlet or outlet blood flows

(5) excision of new growths

(6) intracardiac operations and

(7) removal of pathologic clots in the auricle and auricular appendage and ball valve clots.

In this new field of cardiac surgery will be included the direct attack upon valves *primarily made possible* by the Brock operation the dilatation or fracturing of valves by the surgeon s finger or instrument the insertion of analogous homologous or synthetic valves tissues to close defects and many projected procedures whose realization awaits only the dry chamber in which to operate

### CARDIAC PHYSIOLOGY

A thorough understanding of the cardiorespiratory functions in the body as well as the part any intrathoracic manipulation plays in their malfunction is fundamental to the success of the procedure Of these cardiorespiratory functions several merit special consideration

(*a*) **Anesthesia.**—This subject has been discussed in detail under Anesthesia in Cardiac Surgery (see pages 37 to 40) There are several fundamental points, however, which bear emphasis in connection with heart surgery An *intratrachial tube* should always be utilized whether one hopes to avoid opening the pleura or not The quick return of the reflexes, particularly that of the cough, is of value Some of the anesthetics may cause cardiac irregularities The *accumulation of carbon dioxide* in the body should be avoided [11] Since this gas accumulates in the blood and tissues under normal circumstances, if it is not removed during anesthesia, a severe acidosis may be an early result Thus the soda lime must be changed frequently, be sufficient in amount and of good quality in the closed gas circuit The gas must move between the lungs and the anesthesia apparatus and thus a *good tidal exchange* is required A mechanical compression of the respiratory bag will affect such a change This can be done manually by the experienced An intermittent mechanical compressor has been devised by both Crafoord[7] and Mautz [17] The *anesthetic agent* depends on several factors, but much can be said for ethyl ether which permits the use of high oxygen concentration The skilled anesthesiologist can permit as early a recovery with this agent as with others Cyclopropane is an excellent agent in the hands of a trained anesthesiologist

The type of anesthetic agent varies in different clinics In general, any agent that can produce hypnosis without interfering with adequate aeration is a satisfactory one To some extent the choice depends on the familiarity and experience of the anesthesiologist with the agent Thus Bailey and Glover[12] used pentothal and curare with 2 per cent intravenous procaine To this was added nitrous oxide-oxygen in equal percentage dilution only if needed Harken uses an induction of pentothal and ether He then intubates and carries on the anesthesia with oxygen and ether using the "respiratory assitor" to help respiratory exchange [14] Madden uses straight ether with oxygen and some intravenous procaine [16] In our own Clinic, Collins,[8, 9] our Director of Anesthesiology, uses premedication with a barbiturate (sodium amytal) which has no bronchial constrictor action and an induction with cyclopropane which permits 75 to 85 per cent oxygen to be administered Intravenous procaine (1 per cent) solution is used simultaneously After intubation a "mixture gas" anesthesia is given, the mixture being nitrous oxygen (1 liter), oxygen (1 liter), and cyclopropane (500 cc ) It is of interest that cyclopropane is used by the clinics in which operations are performed on the largest numbers of cyanotic babies [18]

Ruth[1] favors an induction with pentothal and oxygen-nitrous oxide (50 per cent) thereafter. In our experience, the arrhythmias with cyclopropane are prevented by the procaine We believe with Harken that if there is any difficulty with anesthesia, the procedure should be terminated for that day

*Hypothermia* Cooling as an aid or as an anesthetic agent *per se* has had clinical use for some time. This work comes from the application of cooling by Fay to carcinoma patients in 1940 [36] — The early clinical work by Allen and Crossman[1] also showed the value of this modality as an anesthetic agent Our own large series of amputations with this agent

alone in arterial diseases is a matter of record [21] McQuiston[19] working in Pott's clinic uses hypothermia to reduce the anesthesia necessary for the cyanotic children with congenital cardiac malformations Bailey, *et al* [2] followed the work of Bigelow[4] on dogs and showed that with a decreased temperature the metabolism and oxygen requirements could be dropped This increases the time the patient can withstand a cardiac arrest and permits shutting off the superior and inferior vena cavae for from twelve to thirty minutes This closure produces a bloodless right ventricle and auricle We utilize this measure for cardiac and aneurysm surgery to reduce anesthesia permit longer cardiac and brain circulation cessation and to reduce hemorrhage

(*b*) **Blood Volume** —Blood loss is inevitable in thoracic surgery To open the chest will result in the loss of 1000 to 1500 cc [4] The innocuous *loss in the sponges and towels may be most dangerous* Weighing such sponges and towels suggested by Wagensteen [23] is a good guide to the loss The replacement of the blood loss is important both to avoid depletion of the oxygen-carrying capacity of the blood as well as to avoid shock The intra-arterial transfusion has an important place With this method unanticipated sudden losses can be controlled and many of the patients previously lost to hemorrhage or shock can be salvaged In the emergency created by a sudden or overwhelming blood loss the Lord provided the surgeon with the best hemostatic agent his hands There are many patients alive today because the wound of exit was closed by the surgeon's hands The blood loss was replaced intravenous and intra arterial transfusions augmented and the cause for the accidental blood loss then cared for on a patient whose blood pressure pulse and blood volume were near normal rather than in his previous shocked and near-death status Surgical panic must be controlled at such times

(*c*) **Cardiac Arrhythmias** —Since the surface of the heart is irritable its disturbance during operation may result in certain irregularities.

(1) *Ventricular Fibrillation* —This is the most serious of the disturbances If it continues for any length of time, death will result Prophylactically the addition of procaine solution both intravenously (0 1 per cent) and sometimes on the surface of the heart (1 per cent) may reduce the incidence In the presence of ventricular fibrillation intermittent regular compression of the heart manually may break up this fibrillation This compression maintains the circulation temporarily even if the fibrillation continues An electric current passed through the heart has been effective in breaking up fibrillation [3] These electric shocks are given by paddle electrodes and may have to be repeated if the heart fibrillates again Such an apparatus should be in the operating room at all times

(2) *Extrasystoles* —These irregularities occur often during the manipulation of the heart and are inversely proportional to the degree of trauma It is obvious that the heart must be handled carefully displaced as little as possible from its normal position and the manipulation completed rapidly If the heart must be displaced it can be done with a suture through the apex or by traction on the auricular appendage Gentle pressure in such traction yielding with each heart beat, is well tolerated [11]

The role of various other drugs, in the correction of cardiac irregularities has been defined Epinephrine, when injected into the heart, increases the coronary blood flow in a manner similar to the stimulation of the sympathetic nerves This is in contradistinction to the affect of this drug on peripheral vessels where spasm follows its use

## TECHNICAL POINTS IN THORACOTOMY

With modern anesthesiology, blood replacement and antibiotics, as well as improved surgical technics, the thoracic cage can be opened with relative safety today The dynamics of the thoracic cage must be known, understood and constantly kept in mind A tension pneumothorax can kill as quickly as a bullet A hydro- or hemothorax or pericardium requires expert and immediate management Subcutaneous emphysema is best prevented A trained team is necessary if cardiac and thoracic surgery is to be done safely

1 **Incision.**—In general, the incision should center over the point at which most of the technical manipulation will be performed. The surgeon must be cognizant of the change in the patient's position during a lateral or posterior approach, due to extension of the arm or hips, or because of the break in the table In many instances in adults, a rib-spreading operation is easy and less time consuming than a rib resection In younger individuals, a rib resection is safer and technically easier to close Upper thoracic wounds in general are closed easier after rib resection. The increase in the size of the incision by division of the continguous ribs or costal cartilages near their sternal margin is always possible Firm ligation of the intercostal vessels and the internal mammary artery and vein will save much difficulty The sternum may be divided in either direction where needed The clavicle can be excised subperiosteally in part or in toto with impunity Where hemorrhage is feared, an adequate exposure is most important

2 **Lung Collapse.**—With modern anesthesia, the lung can be deflated. Both pleura can be opened at once if it is required The lung should be inflated from time to time and the anesthesiologist should counsel the surgeon in this respect as needed The pleura should not be sutured.

3 **Pericardium.**—The pericardium should be opened whenever the exposure of the part to be operated is incomplete The pericardium may be lightly closed

*Postoperative cardiac tamponade* must be prevented by not closing the pericardium tightly The opening left in the pericardium should not be so large as to permit cardiac herniation Thus small and large pericardial defects are not dangerous, but an opening $2\frac{1}{2}$ inches in diameter may cause serious difficulties [5]

4 **The Heart.**—The normal heart will stand considerable manipulation If arrhythmia should develop, the manipulation should be halted temporarily Control of the irregularities has been discussed An electric shock machine should be available in the operating room to counteract ventricular fibrillation If the heart must be rotated, it is best done by a suture tension

5 **Wound Closure** —The wound must be airtight The sooner the hard walled thoracic cage is restored the more normal will be the intrathoracic dynamics of the heart and lungs The reapproximation of the ribs accurately and simply can be effected by drilling holes near the ends of the ribs A silk suture can be threaded through the drill holes The rib ends may be fitted like a mortice joint The intercostal incision is closed by 5 or 6 pericostal catgut sutures. The muscles are then sutured back into place Such a repair does away *with the avulsed ribs and nonrigid thorax* both of which contribute to leaks tension pneumothorax and emphysema In the upper chest the wound is closed more easily if a rib has been excised If a tube for drainage is not to be left in the chest a catheter should be left in place during the airtight closure This tube should be aspirated to remove all air and blood as the anesthesiologist inflates the lung The tube is then removed and a previously placed mattress suture at the catheter site is tied The subcutaneous and skin closure help to seal the wound Where a tube is to be left it should be connected by tubing to an underwater drainage bottle In case of doubt this latter method always should be used

6 **Postoperative Care** —The success or failure of thoracic surgery depends upon care the patient receives after the operation The house staff must be willing to literally live with the patient for the first day or two thereafter The entire staff must be alerted and briefed on the possible complications their diagnosis and interpretation of symptoms and particularly their treatment Any well trained surgeon can perform thoracic surgery The success or failure and the life or death of the patient depends upon the postoperative attention Unless the surgeon and his staff are prepared to render such care the patient is safer in another surgeon's hands This is true whether the second surgeon is as skilled in the operative technic as the former one because the surgical results are proportionate to the degree and skill of the administered after-care An aspiration set always should be at the bed side Oxygen and expert nursing care must be provided The circulatory system should be kept functioning but not overloaded

## OTHER IMPORTANT POINTS IN THORACOTOMY

1 **Patent Airway** —The patient must have a free access for respiration Bronchial secretions and blood must be removed *Cough*—The cough reflex should be restored as soon as possible as it is Nature's first defense of the open airway The anesthesiologist should have the patient sufficiently awake at the end of the operation for exercise of this function Depressing drugs which abolish the cough reaction are contraindicated *Aspiration*—At the end of the operation the mucous secretions and blood should be withdrawn from the airway by the insertion of a small catheter and aspiration A skilled anesthesiologist can so apply this method as to make it preferable to the bronchoscope The aspiration should be continued until all material is removed and the cough reflex is active *Bronchoscope*—If the aspiration is impossible or unsuccessful the bronchoscope should be inserted and the pulmonary tree cleared Since the instrument is traumatizing its use is restricted to those in whom aspiration fails *Trache*

*otomy*—If other measures fail, tracheotomy is indicated to preserve the open airway In questionable cases this may be the safest measure as the inner tube can be cleaned by the nurse as needed The open airway must be maintained as long as the disease or operation require it, as death will follow an obstruction

2 **Hemothorax.**—Some blood usually accumulates in the chest In general such bleeding follows the insecure ligature of a systemic vessel, the internal mammary or an intercostal vessel If this is increasing it must be controlled The patient may be *x*-rayed sitting up, the chest aspirated and re-*x*-rayed in a similar position an hour later If the blood is increasing re-operation is necessary Shock is no contraindication to re-operation as an intratracheal anesthesia will immediately help the patient by providing oxygenation

3 **Control of Pain.**—Pain is of serious import If there is too much pain a patient will not cough or move around Atelectasis then may complicate the picture For the control of pain procaine blocks are advocated Heavy narcotics are dangerous Mild doses of the barbital drugs help greatly in pain control and are best tolerated by these patients Abdominal symptoms including gastric distention are a complication of pleural pain. For these symptoms an intestinal tube with aspiration is advised.

4 **Re-expansion of the Lung.**—In an emergency with a nonexpanded lung, the chest should be tapped and aspirated If there is respiratory embarrassment a tube should be inserted and connected to under water drainage If the lung is not re-expanded in a week, it is likely that a clot or fluid collection is maintaining its collapse This must be removed by aspiration or thoracotomy if required

## CARDIAC DISEASE DIAGNOSIS

The standard diagnostic methods for determination of the cardiac anomalies, malformations, injuries, diseases and dysfunctions include the *x*-ray, the electrocardiograph, the serial kymetrograph and teleoroentgenograms, the contrast *x*-ray technics, etc These latter include barium swallows, contrast dyes in the trachea and pulmonary tree, aortograms, angiocardiograms and cardiac catheterization The multiple film technic permits serialization of the films similar to those seen in moving pictures, and with correct timing the relation of various parts of the cardiovascular system to their normal and abnormal functions can be discerned Catheterization permits pressure and oxygen content determinations made in the various chambers of the large vessels and the heart itself To these methods has been added the direct exploration of the heart itself In the questionable cases it is a logical and fact-finding operation The heart, its coverings, its large vessels and now its interior can be examined This is not to be considered a mere diagnostic procedure as at the same time definitive reparative surgery can be undertaken Thus, one may use the direct examination for

1 Palpation of thrills and listening of bruits, valuable in pulmonary stenosis

2. Dilatation of pulmonary artery or other vessels distal to an obstruction.

3 Hypertrophy or atrophy of parts of the heart indicating obstruction atresia or overuse

4 Narrowing or constrictions of parts of the heart denoting septal defects.

5 Stricture or narrowing as seen in the pulmonary artery or in coarctation of the aorta

6 Absence or transposition of sections of the heart

7 Reduced ventricle and enlarged auricle (as seen in mitral stenosis)

8 Enlargement of the pulmonary veins and atrium due to mitral stenosis

9 Thrills diagnostic of lesions of heart *i e* diastolic over L ventricle apex (mitral stenosis or aortic regurgitation) diastolic over L ventricle base systolic over L atrium (mitral regurgitation) diastolic over R ventricle apex (tricuspid regurgitation) systolic diffused over R ventricle base (infundibular stenosis) systolic over pulmonary artery (pulmonary stenosis) and systolic over aorta (aortic stenosis)

These tactile measures may be made more definite by intracardiac palpation which has its place to confirm diagnoses before definitive surgery is performed and at the time it is done Thus palpatory cardiac diagnosis can be used as a confirmatory test at operation time

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Chapter

8

# SURGICAL TREATMENT OF WOUNDS OF THE HEART

WOUNDS of the heart may be caused by penetrating and nonpenetrating injuries. While most such wounds are of the perforating type with a laceration of the pericardium and its contents, over 160 traumatic heart lesions due to nonpenetrating injuries have been reported in the last twenty years.[14] The discussion of these wounds will be divided into the penetrating and nonpenetrating types.

## PENETRATING WOUNDS OF THE HEART

The day when a wound of the heart automatically meant death has passed. Many of our past ideas of automatic death after a cardiac injury were due to associated pneumothorax, tension pneumothorax and hemothorax. The danger and gravity of a cardiac insult is not depreciated but the possibility of the survival after such an accident needs emphasis. The surgeon who sees these patients first is usually the untrained interne on the ambulance or in the accident room. Emergency hospitals must be prepared for the standardized methods necessary if the patient is to survive.

**Etiology.**—The most common causes for penetrating wounds of the heart are gunshot or stab wounds. In addition to war, homicidal or suicidal attempts, rapid travel with its incidence of trauma, flying metal and glass is an increasing cause. Most such wounds are over the cardiac silhouette and if penetrating are suspicious. Wounds in the axilla and above the clavicle frequently open into the pericardium. The incidence of cardiac wounds at Harlem Hospital in New York showed that 7 per cent of the chest wounds entered this organ.[14]

**Symptoms.**—Approximately 1 in 4 patients reaching a hospital after a penetrating wound of the heart are moribund. Those with myocardial injuries are always in shock, while if only the pericardium is injured, one-half present pre-fatal signs. This shock is often, but not necessarily, of prognostic significance.

According to Bigger's[4] classification, heart wounds are divided into four types. Those in Group 1 are considered minor and their symptoms depend upon the amount of blood loss. Hemothorax and shock when present are mild. Group 2 and Group 3 have symptoms of tamponade. In those in which the tamponade is stationary, the initial shock usually decreases. This may be due to the cessation of bleeding as occurs if a laceration seals or clots itself, or it may be due to the evacuation of the blood collection from time to time into a coexistant pleural wound or externally. In the

latter group, subsequent shock may develop. In Group 3, the tamponade is progressive with diminishing cardiac sounds, increasing cardiorespiratory embarrassment and eventual cardiac standstill In this classification, a wound injuring a coronary artery would be in Group 2 or Group 3. The symptoms of such a wound would be those of myocardial infarction. In Group 4, exsanguination is the principle symptom There may be serious hemothorax and many of these patients die before any therapy can be extended

**Symptoms of Tamponade.**—Shock is predominant The skin is cold and moist and there is cyanosis The heart sounds are weak, irregular or inaudible as is the pulse The blood pressure is low or zero The venous pressure is elevated This is indicated by dilated veins in most cases

**Pathologic Physiology.**—In a patient with a normal heart, the right atrial pressure is 30 to 31 mm of water [24] The blood is returned to the right heart because the pressure in the vena cava is greater than that in the right auricle When there is an increased pericardial pressure, the blood entering the right heart is backed up, the vena cava pressure rises and there is a damming back of all the venous blood At the same time, the arterial pressure drops with hemorrhage and with the venous and arterial vasoconstriction. Thus, there is a decrease in the blood volume on the arterial and an increase on the venous side As the tamponade increases, the atrial pressure rises and this may be over 250 mm of water This pressure is too great for the venous pressure to overcome and the circulation fails When this is an acute change, and the pericardial effusion develops rapidly, death occurs When the effusion is gradual in onset, the pericardium dilates and becomes thinner and thus gives some relief to the atrial pressure In the latter instance, larger amounts of tamponade are tolerated In like manner, if the pleura is opened by the wound, the fluid around the heart can leak out and thus relieve itself An understanding of these basic principles involved, will make the management of such a patient more simple The mechanical pressure of the tamponade may stop cardiac contractions [7,25]

More recently some experimental work has shown that stabs or lacerations of the heart muscle are not fatal most of the time unless the coronary vessels are injured Dogs' hearts survive repeated stab wounds even with tough instruments such as scissors and even where the pericardium is sewed up thereafter, the heart often seals off the laceration . again providing the coronary vessels are inviolate A small injury to the latter, however, causes cessation of the heart contraction [7,25] While the carry over to the human may not be exact, our concepts on the fatal termination of tamponade may have to be changed

Since the first successful suture of a stab wound of the heart by Rehn in 1896,[22] the therapy in this group has steadily improved While the mortality in 1909 (160 patients) was 64 per cent,[20] Elkins reported a reduction to 22 per cent in 1944 [11] The combination of aspirations and intravascular fluids has further reduced this mortality [12] In this respect, intra-arterial blood transfusion has a particular value because of the large quantity of blood that can be given rapidly This arterial transfusion provides a volume against which cardiac systole can exert a pressure.

**Treatment.**—A. ASPIRATION AND TRANSFUSION.—If intravenous injections are used, a positive pressure should be applied to speed the flow. In the presence of cardiac tamponade, a higher intracardial pressure can be withstood by the patient with the installation of intravascular fluids.[5,10]

If the heart wound is small, supportive therapy alone or with aspiration of the pericardium may be sufficient to save the patient's life. In many patients with small heart wounds, the wounds are held open by the surrounding tamponade. In such cases aspiration of the tamponade and rapid intravenous or intra-arterial fluid therapy have so restored the cardiac contractions that the wound is closed by the contractions or locked by the clot. Whether to operate or not must be decided in each case. If the patient's condition is improving and the patient comes out of shock, one needs to add no further therapy. If the shock continues or recurs, or if aspiration fails, surgical intervention is the only hope. Aspiration should be performed in each case but during this time preparations for operation should be completed. One must be ready to continue the surgical procedure if attempts to relieve the tamponade fail, shock recurs, or the blood repeatedly accumulates.

The value of the aspiration technic was well shown by the mortality rate of 5.9 per cent reported by Elkin.[12] The patients who do not respond to aspiration probably have associated serious injuries such as wounds communicating with the chest or abdomen. This aspiration technic restores the dynamics of the cardiovascular circulation. Its success depends upon aspiration before the blood has clotted. In those wounds which continue to bleed, aspiration must be repeated. If this technic is used, it must be remembered that a dry or poor tap result in the presence of a known tamponade dictates an immediate exploration. If the aspiration is dry, observation is indicated for the "slow leakers" and they should be followed clinically and by roentgen ray for several days. The idea that cardiac tamponade is not fatal necessarily is not new and was advanced in the 16th Century.[4,14] Morgagni[19] first suggested the part that tamponade played in death. The medical profession unanimously condemned any attempt to enter the pericardial sac. Dupuytren, the surgeon to the Duc de Berry, the heir to the French throne, kept his patient alive after he had been stabbed in the heart, by introducing a probe into the pericardium for some time. He was reprimanded.[4] The first attempt made at aspiration was by Larrey in 1829,[17] who followed the suggestions of Riolanus.[22] The high mortality of 84 per cent reported in 1868 by Fischer[13] probably was due to the associated venous section then in vogue. The re-evaluation of this therapy led to its acceptance.

*Technic.*—Five per cent glucose is started intravenously at once. An intra-arterial cut-down is prepared and blood substituted for the glucose. Other injuries are ruled out by physical examination and roentgen ray. A pericardial aspiration is made with a 17 gauge needle inserted through the left fourth intercostal interspace. If this is not successful another aspiration is made as follows. The patient is placed in a prone position and a needle is inserted in the epigastrium just to the left of the xiphoid process with the needle directed superiorly and fairly superficially (just below the costal cartilage). Suction is maintained on the syringe plunger

A dark liquid fluid will be obtained when the pericardium is entered The aspiration is continued while liquid can be obtained and repeated if the patient relapses into shock (See pericardial tap, page 151)

*Sequelæ* — Pericarditis or pericardial effusion is possible but an unlikely complication It can be treated by aspiration A suppurative pericarditis occurs only in the infected patient Antibiotic therapy will prevent most such complications

B Operative Treatment —If operation is elected, it must be done at once Similar to cardiac arrest, one cannot wait for anesthesia, boiled instruments or sterile drapes The mechanical relief of the tamponade often will be lifesaving if supplied in seconds or minutes The hemorrhage from the heart wound can be controlled manually or secondarily repaired

An incision is made transversely from the sternum to the left nipple line, in the fifth interspace or the space below the stab wound.[10] The muscles are divided rapidly, the ribs are spread and the costal cartilages are cut The pleura is avoided if possible In this exposure, the Finnochietto rib retractor is valuable If the heart is beating and the wound can be seen, it may be sutured before the removal of the tamponade The right ventricle is the most common site of the injury

The hemorrhage from the wound site will be severe when the clots are removed A finger introduced into the wound site, if it can be seen, will help control the bleeding If this is not possible, a finger should be pressed above and below the probable wound site A suture rapidly placed in the heart muscle above and below the fingers acts both as traction and as a hemostatic suture A stay suture may rotate the heart into a better position for suturing This is helpful when the wound is in an inaccessible site If the wound is near the coronary vessels, one should identify them and avoid placing a suture near them as they are necessary for life

Extrapericardial exsanguination is the cause of death in most patients who do not survive long enough to reach the operating table Aspiration will remain a safer procedure as a primary step It may be the elected treatment in hospitals not equipped for immediate intervention.

The control of wounds of the ventricles and auricles of dog hearts without suture by the application of a gelatin sponge may be revolutionary These gelatin sponges have been used even to stop hemorrhage from the wounds in coronary vessels in experimental animals They are applied by the "patch method" and have been effective in stopping bleeding from the auricle, the ventricle and the liver[15] The possibility of introduction of a hemostatic agent through the aspiration needle or by a cardioscope exists

**Treatment of Cardiac Wounds.**—The following regimen is recommended in management of wounds of the heart

1 Institute positive pressure intravenous infusion This may be started with glucose or plasma and followed with blood At the same time, an intra-arterial cutdown infusion is begun

2 Administer oxygen by respirator Be sure airway is open Use positive pressure

3 Close sucking wounds by suture or pack

(*a*) Aspirate air from chest cavity for tension pneumo- or hemothorax

(*b*) Stop obvious bleeding wounds,

4 Aspirate the pericardium

5 If there is evidence of increasing tamponade bleeding or if the patient relapses into shock an immediate operation is necessary The same is true if there is a dry or inadequate tap

6 In such event open the chest through a transpleural thoracotomy at the fourth intercostal space Spread the wound with a rib spreader Open the pericardium avoiding the heart which may be pressed against the pericardium by the tamponade [21]

Hold the wound with the fingers or hand or with traction sutures above and below it Suture the wound and evacuate the clots Close the pericardium partially Close the chest in airtight manner leaving an intrapleural catheter for underwater drainage

7 Continue shock therapy

8 Antibiotics and sometimes antithrombotic therapy are indicated

## NONPENETRATING WOUNDS OF THE HEART

The fact that the heart could be injured by nonpenetrating wounds was recognized as early as 1764[1] Only 27 such injuries were reported in the two hundred years up to 1868 [26] It is now recognized that 15 percent of all fatal chest injuries will be due to a nonpenetrating trauma The machine age and more rapid travel have raised the incidence of this type of heart injury The steering wheel injury is an example of the cause of contusion of the heart

**Symptoms.**—The condition is more often missed than diagnosed because its possibility is not considered *Pain* is the most frequent symptom and this varies with the type of lesion produced Trauma to the pericardium causes pain in respiration while that of the myocardium simulates the pain of a coronary occlusion A ruptured valve causes a murmur The injury to chordae tendineae produces a vibratory bruit Many of these symptoms are masked by shock or other injuries The arrhythmias vary with the types of injury Hemopericardium causes more distant heart sounds and distension of the neck veins X ray examination of the heart and mediastinum will aid in diagnosing the lesion Kymoroentgenography and electrokymography may localize the lesion To differentiate the condition from coronary artery occlusion may be difficult and often depends upon the history Pericardial tap should be performed in any doubtful case and where signs indicate the development of tamponade

**Pathology**—The pericardium is most often the injury site Subpericardial hemorrhage occurs in most of the patients Such lesions develop from compression against the spinal column Crushings or falls from a high place may cause a "bursting" injury A bursting may occur from compression of the great vessels and their re-expansion in which event the wound is larger on the outside than on the inside The myocardium may be lacerated as may the tendineae which hold the valves while the cusps are bent by the cardiac compression The pulmonary and tricuspid valves rarely are ruptured

**Prognosis**—The prognosis depends upon the degree of injury. Mild myocardial and pericardial contusions tend to heal. More severe ones result in fatal arrhythmias. Symptoms of coronary disease may persist. Injuries to valves cause congestive failure and usually death. The pericardial lacerations may be followed by tamponade. In event of survival, adhesions may develop. Embolism is always a possibility.

**Treatment.**—In the patients with shock, pericardial tap to relieve the tamponade and as an aid in diagnosis is indicated. If cardiac fibrillation occurs, electrical stimulation should be used. If not available, an intracardiac injection of adrenalin may start the organ. Cardiac massage may start the heart, but usually is only temporarily successful. If the patient survives, the therapy should be the same as used for that of coronary artery occlusion. Two to five weeks of bed or chair rest is suggested. For anoxia, oxygen should be supplied. Sedation is necessary. The arrhythmias tend to revert to normal rhythm. If auricular fibrillation develops, quinidine therapy may break it. Pronestyl and quinidine may avert these arrhythmias. The surgical repair of the ruptured cardiac muscle should be considered if the patient survives the original lesion, but still shows tamponade or a tendency to relapse into shock. The prognosis surgically is serious.[9]

In the treatment of these lesions, the occasional patient will be misdiagnosed. An acute coronary occlusion may be operated. In such event, an effort to remove the clot in the coronary artery is indicated.

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Chapter

# 9

# SURGICAL TREATMENT OF CONGENITAL HEART LESIONS

*Pulmonary Stenosis; Patent Ductus Arteriosus; Coarctation of the Aorta, Anomalies of the Arch of the Aorta and Its Branches; Other Cardiac Anomalies—Septal Defects*

**Classification of Congenital Heart Disease.**—The technic of correcting congenital malformation of the heart and surgeons capable of performing these procedures have been added to so many hospitals in the last few years that one forgets how recently these surgical methods were perfected. While Carrel and Tuffier[33] had attempted to incise a stenotic valve in 1924, the first successful operation for the tetralogy of Fallot was performed by Blalock in 1944.[21] The first operations for coarctation of the aorta were performed the same year by Crafoord[37] of Sweden and Gross[66] in Boston. These three men changed congenital cardiac surgery from a potential to an actual fact.

Our knowledge of congenital heart disease began with Maude Abbott's[5] monograph twenty years ago. Taussig's outstanding clinical evaluation of congenital heart disease was the next major advance in diagnosis.[123] Gross's ligation of a patent ductus in 1938 was the first successful correction of a cardiac defect.[57,62]

Congenital heart disease may be classified on a clinical, physiologic or pathologic basis. Abbott's original classification was based on the presence or absence of cyanosis.

*Abbott's Classification of Congenital Heart Disease*[4,5]

1. Acyanotic Group (no abnormal communications)
2. Arteriovenous Shunts Group (have terminal reversal of flow)
3. Cyanotic Group

*Taussig's Classification of Congenital Heart Disease*[123]

1. Malformations which reduce the oxygenated blood below that sufficient for growth.
2. Malformations in which sufficient oxygenated blood is supplied for growth.

Since surgical intervention depends upon physiologic and not anatomic changes, the classification of Bing seems most logical (see p. 73).[12]

**Diagnosis.** The diagnostic points in congenital heart disease will be taken up under each lesion. The importance of an accurate diagnosis cannot be overstressed. An error may subject a child to a needless and possibly fatal operation. In the diagnosis, the *history* is of great importance. The onset of cyanosis, its severity, the degree of dyspnea, squatting, the reaction to exercise, unconsciousness and convulsion must be carefully

CLASSIFICATIONS OF CONGENITAL HEART DISEASE[12] (BING)

I *Pulmonary Flow Less than Systemic Flow* (Pulmonary Artery Pressure Usually Decreased)
1) Tetralogy of Fallot
2) Pseudotruncus arteriosus
3) Tricuspid atresia
4) Single ventricle
   a with pulmonary artery arising from a rudimentary outlet chamber
   b with pulmonic stenosis
5) Transposition of the great vessels with pulmonic stenosis
6) Patent foramen ovale with pulmonic stenosis
7) Ebstein's disease with patent foramen ovale
8) Anomalous venous return
   a. pulmonary arteriovenous fistula
   b superior vena cava emptying into the left auricle

II *Pulmonary Flow Greater than Systemic Flow* (and/or Pulmonary Artery Pressure Normal or Increased)
1) Eisenmenger's complex
2) Transposition of the great vessels
   a complete
   b partial
3) Isolated septal defect
   a auricular septal defect
      1 uncomplicated
      2. Lutembacher's disease
   b ventricular septal defect
4) Aortic atresia with patent ductus arteriosus
5) Single ventricle
   a. with both great vessels arising from the rudimentary outlet chamber
   b with the aorta arising from the rudimentary outlet chamber
6) Truncus arteriosus
7) Patent ductus arteriosus
8) Anomalous venous return with pulmonary vein emptying into the vena cava or the right auricle

III *Pulmonary Flow Equals the Systemic Flow* (at Rest and After Exercise)
1) Pure pulmonic stenosis
2) Patent foramen ovale as an isolated anomaly
3) Coarctation of the aorta
   a. adult type
   b infantile type without patent ductus arteriosus
4) Double aortic arch

*Definitions* Systemic Flow equals venous blood entering right auricle pulmonary artery flow equals volume of blood entering lung by pulmonary artery alone Note Systemic flow may be exceeded by aortic flow in a shunt *i e* patent ductus arteriosus

elicited Where the family history is unreliable, the patient should be observed sufficiently long to obtain such data The *physical examination* should include the habitus, the development, cyanosis and clubbing. The blood pressure in all four extremities, the apical thrust, the size of the heart, the position of the great vessels, the size of the liver and spleen and pulmonary congestion are all significant The thrills and murmurs may be diagnostic when considered with the other findings *Fluoroscopy* and *roentgenography* are of great importance In the anteroposterior position, the right border of the heart is made up of the right auricle and the superior vena cava Enlargement of the left side of the heart rotates the heart and accentuates the aortic nob The left anterior oblique position permits comparison of the right and left ventricle [9a] The right anterior oblique position presents the right auricle and ventricle, the ascending aorta and the pulmonary artery The left auricle is against the esophagus and its enlargement can be determined by a barium swallow *Electrocardiographic* readings may be of some value. Physiologic studies are used when necessary to confirm a diagnosis and to demonstrate how the patient is coping with his anoxemia. These studies include

(*a*) angiocardiography,
(*b*) the exercise test,
(*c*) oximetry which registers variations in the oxygen saturation in the body,
(*d*) pulmonary capillary flow, which measures the total blood flow through the lung,
(*e*) catheterization of the heart

## PULMONARY STENOSIS

### PURE PULMONARY STENOSIS, INFUNDIBULAR STENOSIS—THE TETRALOGY OF FALLOT

Pulmonary stenosis is the most common congenital heart lesion in the cyanotic group More than three-fourths of all cyanotic cardiac patients have pulmonary stenosis Since these lesions are congenital the abnormality may vary greatly Any degree of the classical picture may be present The stenosis usually involves the musculature of the right ventricle below the valve in the infundibulum of the right ventricle. More rarely, the stenosis involves only the pulmonary valve[121] (pure pulmonary stenosis)

The tetralogy of Fallot is characterized by pulmonary stenosis, an interventricular septal defect, an overriding aorta and hypertrophy of the right ventricle As a result of the deformities, blood from both ventricles enters the aorta which is over the opening in the septum Two physiologic disturbances occur The first is the mixture of oxygenated and unoxygenated blood which is thus distributed to the peripheral circulation due to the *dextroposition* of the aorta [20] No satisfactory surgical solution for this problem has been found as yet, although animal experimentation makes it appear that some such defects can be corrected The second pathologic result is due to the *stenosis* of the pulmonary artery As a result, the amount of blood entering the lung for aeration is inadequate Systemic anoxemia follows For this latter condition surgical therapy has been employed successfully

**Symptoms.**—The most common symptom is *cyanosis* The cyanosis usually begins in the first year of life and increases as the child becomes more active. The cause for the cyanosis is the insufficient number of oxygen carriers being aerated by the lungs The importance of the interchange of oxygen by the lung and the direct correlation between the number of oxygen carriers reaching the lung and the degree of cyanosis has been established [illegible] The children *squat* after exertion *Dyspnea* is severe and occurs with the cyanosis The children are stunted in growth and walking is delayed *Clubbing of the fingers* is usual The degree of clubbing depends on the length of time that the pulmonary stenosis has existed *Polycythemia* develops The red cell count may be as high as 8 or 9 million and a hemoglobin of 24 grams is not unusual The hematocrit reading is high Peripheral thrombosis follows in multiple areas The *oxygen saturation* in the arterial blood will be low and may be only 30 to 40 per cent of normal

*Neurologic lesions* may follow the areas of thrombosis Even hemiplegia and aphonia may be seen The *electrocardiographic* tracings are not diagnostic but show a right axis deviation and the P waves usually are high

**Diagnosis.**—The diagnosis of pulmonary stenosis is made mainly on the symptoms *Cyanosis* is the principal sign A systolic thrill and murmur along the left border of the sternum is a frequent but not necessary finding With the *fluoroscope* the contour of the heart is characteristic In the anteroposterior position there will be a concavity in the pulmonary conus In the left anterior oblique position the heart is enlarged anteriorly In the right anterior oblique position a concavity will be seen at the junction of the aorta and the right ventricle In one-fourth of all the patients, the aortic arch will be on the right side. This can be determined by the barium swallow as the aortic arch will indent the esophagus on the side on which it lies This has surgical significance in choosing the operative approach

If one elects the Potts operation or some modification of it prior knowledge of the location of the descending aorta becomes imperative in order to open the chest on that side While angiocardiography clearly demonstrates the aorta cyanotic infants especially under one year of age withstand such a procedure poorly Angiocardiography has a high mortality in this group In questionable cases the insertion of a polyvinyl catheter through a needle in the femoral artery and its passage to the aorta has been an aid in localization of the aorta A small amount of contrast material outlines the course of the catheter without delineating the aorta itself This method has been a diagnostic help [137]

*Cardiac Catheterization* — Cardiac catheterization is not necessary or desirable in all patients It is an aid in diagnosis but is not without its dangers Its use should be limited to those patients in whom a confirmation of the diagnosis is required Experience is necessary in order to interpret the results of this procedure

(*a*) The movement of the catheter may indicate the degree of stenosis The catheter has been passed through the stenosed valve The septal defect is shown when the catheter moves into the left ventricle [illegible, 40]

(*b*) The *flow* through the pulmonary artery can be determined also by the catheter In most, the pulmonary artery flow is below normal

(*c*) The *pressure* in the cardiac chambers and in the pulmonary artery can be determined The pressure in the right ventricle will be elevated. (from 40 to 110 mm above normal) The ventricular pressure rises and falls with systole The ventricular diastolic pressure is above normal The pulmonary artery pressure is low The relative pressure between the right ventricle and pulmonary artery is significant [12,14]

*Pulmonary Capillary Flow* —Pulmonary capillary flow will be greater than the pulmonary artery flow indicating collateral circulation The

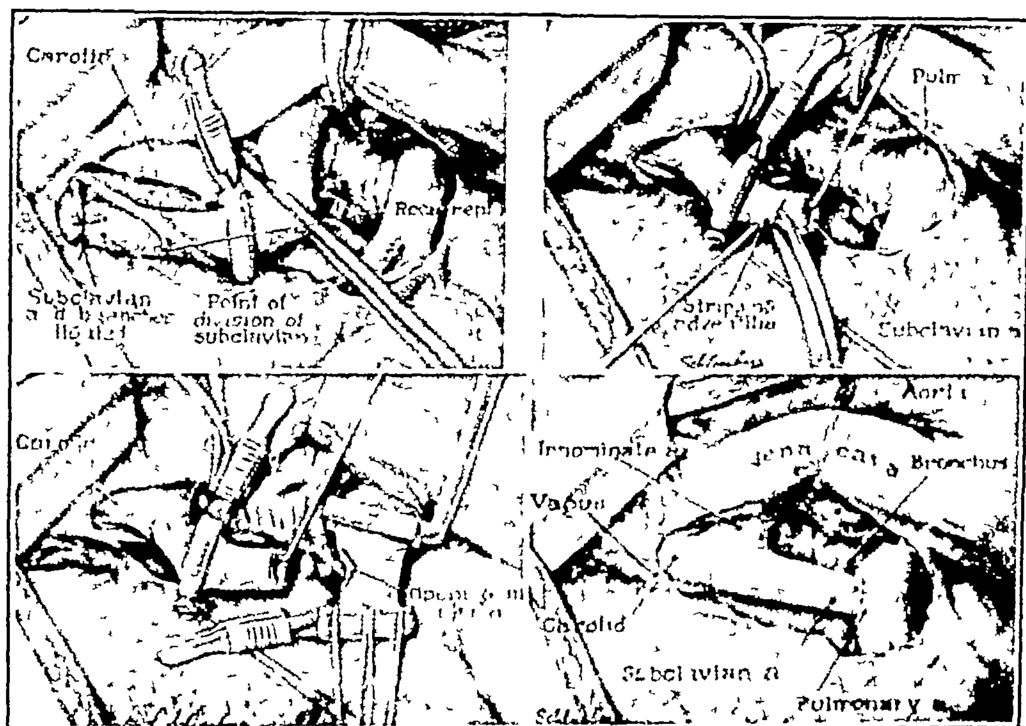



Fig 8 —Technic of Blalock operation for pulmonary stenosis Subclavian artery anastomosed to pulmonary artery end to side fashion (Courtesy of Doctor A Blalock and permission of Surgery, Gynecology and Obstetrics )

pulmonary capillary flow can be determined by the analysis of $CO_2$ in expired air. Comparing the pulmonary capillary and pulmonary artery flow gives an estimate of volume flow [12,14]

*Exercise Test* —The ratio of oxygen consumed per liter of ventilation decreases when the patient exercises from what it was at rest (87 per cent).[14] This fall is due to the limitation of the volume of mixed venous blood which can circulate through the lungs during exercise If this fall does not occur, the lesion is minimal or there is an adequate collateral circulation. In a right to left shunt, this decrease in oxygen per liter of ventilation is associated with a reduced oxygen saturation of the peripheral arterial blood [12]

*Angiocardiograph* - The diodrast will enter the right ventricle in two seconds The aorta and its branches will be outlined Usually, the pulmonary artery and its conus will not be outlined. The pulmonary artery may fill slowly by collaterals

**Treatment.**—In the tetralogy of Fallot the stenosis most often is in the infundibulum The pulmonary valve usually is normal The aorta overrides the interventricular septal defect This malformation may be treated by a recirculating operation It can also be treated by a direct attack upon the stenosed area as shown by Brock [18,25] The pure pulmonary stenosis in which the valve has stenosed is a separate entity and requires a different surgical approach if right cardiac failure is not to follow In most such patients a patent foramen ovale will persist because it is held open by the raised pressure of the right auricle Two types of operations have been devised—the recirculating and the valvulotomy operations

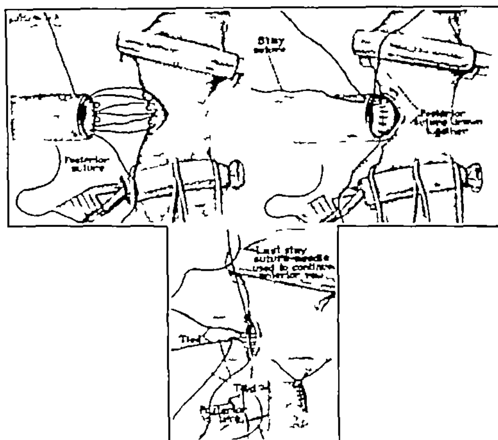



Fig 9 —Technic steps in anastomosis of subclavian artery to pulmonary artery Sutures are of five-O Deknatel silk everting, interrupted and through entire wall of artery This suture everts the intima. Guy sutures line up the anastomosis. (Blalock, courtesy of Surg. Gynec and Obst )

A *Recirculating Operations* —Three recirculating operations have been described If possible the operation is delayed until the child is between four and eight years of age This makes the diagnostic tests safer and the operation more simple as the vessels have been enlarged In some children the lack of oxygen so severely handicaps them that operation must be performed earlier

Blalock Operation —Blalock devised an artificial ductus arteriosus for the treatment of patients with pulmonary stenosis. The side on which the aorta descends is determined (see page 7[illegible]) and the incision is made on the side of the chest *opposite* the side on which the aorta descends This is important since in his operation the subclavian artery is used as it

comes off the innominate artery. The innominate artery is always on the right when the aorta descends on the left, and vice versa

The subclavian artery, because of its availability and size, is used. The subclavian artery on the side on which the innominate artery arises makes a better angle for anastomosis than when the subclavian artery arises from the aorta [17, 18]

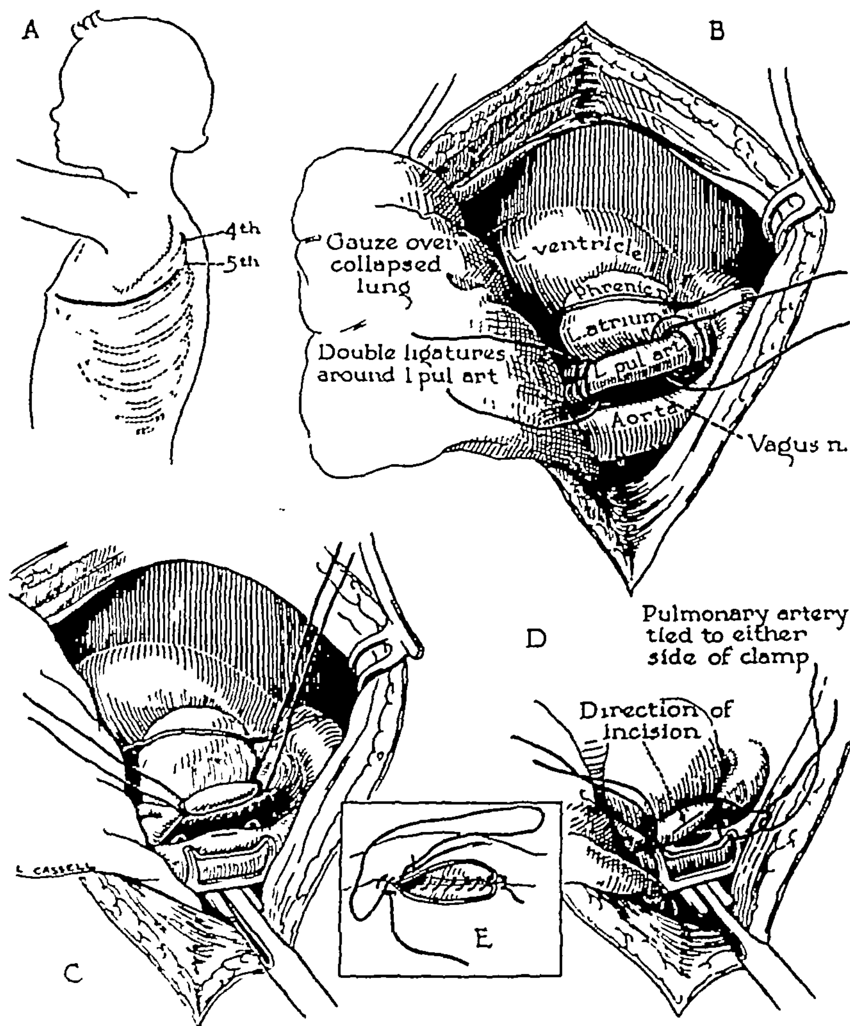



FIG 10 —Potts' technic to relieve pulmonary stenosis

A, Incision B, Dissection of pulmonary artery and aorta C, Occlusion pulmonary artery Incision in aorta after application Potts' Clamp D and E, Completion of procedure (Courtesy Dr W Potts, Chicago)

The pleura is opened and the pulmonary artery is exposed This requires resection of the azygos vein The pulmonary artery is dissected free to attain the length sufficient to permit anastomosis If the pulmonary artery pressure is greater than 350 mm of water, an anastomosis should not be performed

The differentiation of the pulmonary artery and vein may be difficult and in questionable cases the pericardium should be opened Should the pulmonary vein be selected instead of the pulmonary artery in error, pulmonary edema and death would result from the anastomosis The

pericardium should be opened also if it is difficult to obtain a sufficient length of pulmonary artery The innominate artery is then freed after dividing the mediastinal pleura This vessel must be dissected for a considerable distance to give sufficient length for an adequate anastomosis If necessary the carotid artery is divided to permit the innominate and subclavian arteries to be mobilized The subclavian artery then is divided and ligated at the point where it gives off its first large branch In a few instances when the subclavian artery technically cannot be mobilized sufficiently, Blalock has used the common carotid artery for the anastomosis, but the attendant danger of hemiplegia in 10 per cent of the cases when the common carotid artery is divided then must be accepted and faced

The identification of the main pulmonary artery must be made carefully as the pulmonary artery to the right upper lobe may be mistaken for the main one The end of the systemic subclavian artery is anastomosed to the side of the pulmonary artery this technic being preferable to the end to end anastomosis.

The suturing is done with fine silk the sutures everting the intima The sutures are placed to bring the intima to intima without the interposition of the adventitia The sutures should not be placed too tightly as the pressure at the suture point decreases when the lung is inflated and the instruments removed from the chest (See Figs 8-9) All of the technical points mentioned in general cardiac surgery anesthesiology opening and closing the chest apply to operations on these congenital heart patients The closure must be airtight A catheter is left in the pleura during the closure It is aspirated at the end of the operation as the anesthesiologist inflates the lung It is safer to leave the tube in the pleura and connect it to underwater drainage to assure against the development of a tension pneumothorax The postoperative care requires the careful observation of the patient for complications and their prompt treatment if they develop The postoperative care has been detailed on pages 61 and 62

Potts Operation —Pulmonary stenosis has also been relieved by recircuiting after the Potts method Potts operation consists of a lateral anastomosis between the left pulmonary artery and the thoracic aorta This operation technically is easy to perform in patients past twenty years of age and the size of the shunt can be made as large as it is desired Potts ingenious clamp which permits some aortic blood to flow even while the suturing is being performed is an important addition to the technic (Figs 10 and 11) The clamp has been adapted to many other operations The technical points outlined under the opening and closing of the thorax anesthesia and after care have the same adaptation in this operation but are not unnecessarily repeated

Holman s Operation —*Holman s operation*[71] consists of the use of the subclavian artery on the side *opposite* the innominate artery where the subclavian artery comes directly off the arch of the aorta The objection of Blalock that this artery may kink has been corrected by Holman When the subclavian artery does kink, Holman divides the pulmonary artery on the heart side of the anastomosis thus correcting the kinking The Holman s operation at times is technically more simple than either of the other methods of anastomosing Since these operations are performed

on children whose vessels are expected to grow, the suturing should be of the interrupted mattress type In experimental animals, this permits the vessel to increase in size with the maintenance of the shunt [116]

B *Valvulotomy for Pulmonary Stenosis* —THE BROCK PROCEDURE —The possibility of a direct attack on the stenosed valve has been considered for some time, but the lack of positive pressure anesthesia, the incidence of infection, hemorrhage, and fear prevented a trial of such a procedure for many years The first successful suturing of a heart by Rehn[45] in 1897 proved that the heart was an organ which could stand surgical intervention

Doyen[46] was the first to attempt to divide a stenosis which he considered to be valvular in 1913 The patient died and the autopsy showed that the stenosis was below and not at the valve Doyen used a tenetome knife introduced through the right ventricle Cutler and Beck[38a] subsequently tried to divide mitral stenotic valves

While the work of Blalock and the others who perfected short-circuiting operations was revolutionary and decreased the cyanosis and disability, certain objections to this attack were obvious The pulmonary obstruction remained, an artificial ductus arteriosus was added to an already abnormal heart and eventually, as the pulmonary stenosis increased with clotting, fibrosis and contraction, the patient was dependent upon the anastomosis entirely for oxygenation [24] In addition, some of the shunts closed [84] Brock achieved the first successful results in the direct approach to relieve the stenosis Brock,[26] in 1948, reported on three operations in which the pulmonary valve was divided directly His operation was used originally when there was a valvular and not a subvalvular or an infundibular stenosis He now uses this operation for the tetralogy of Fallot He has done over 30 such operations and has selected the procedure in 45 per cent of the last 50 patients with this lesion in preference to the Blalock or other recirculating procedures [24a,25,25a]

VALVULOTOMY FOR PURE PULMONARY STENOSIS —The diagnosis is difficult to make It is confused also with Fallot's tetralogy It can be differentiated by the clinical picture and angiocardiography in most instances A poststenotic dilatation of the pulmonary trunk with delay in emptying of the contrast medium is a feature [23] This is not so common in the tetralogy of Fallot because the pulmonary trunk is smaller and the overriding of the aorta carries away most of the contrast media Brock states that if an infundibular stenosis cannot be shown by angiocardiography, pure valvular stenosis should be suspected

In the pure pulmonary stenosis a recirculating operation not only fails to relieve the right ventricle but increases the burden on the left ventricle Even though cyanosis may be relieved, the danger of heart failure is increased In such cases, congestive failure follows and the life of the patient can be saved only by relieving the pulmonary stenosis If, under a mistaken diagnosis, a systemic pulmonary anastomosis has been made, this must be undone In the valvular stenosis, the valve is closed by a septal fusion with a small projection into the pulmonary artery through which the blood is ejected in a thin stream Brock believes that external examination of the exposed heart will tell the type of stenosis present.

Some diagnostic points in *valvular pulmonary stenosis* are

1 The stem of the pulmonary artery distal to the obstruction is dilated forms an aneurysmal-like bulge visible through the pericardium and has a characteristic thrill

2 If the sinuses of Valsalva are present and formed it indicates the valves are also present and normal When a stenosis is present there is a dilated artery above the valve level or an aneurysmal dilatation exists in the pulmonary artery If the dilatation is slight it suggests a subvalvular stenosis

3 Palpation of the first part of the pulmonary artery shows a high pitched thrill localized just above the origin of the artery and this can be felt to be localized in the center of the lumen

4 One can feel the conical valve between heart beats in valvular stenosis

5 The thrill fades out rapidly on the right ventricle wall

In this pure valvular stenosis the correct treatment is the relief of the valvular obstruction by direct valvulotomy If an anastomotic operation is performed right-sided heart failure eventually results This is due to the fact that the already overworked right heart is further overloaded by an added systemic blood flow in the lungs

In *subvalvular* stenosis 1 There is no localized thrill 2 The thrill is coarse, low in frequency felt best below the valves 3 Direct valvuloscopy through the ventricle has been used to determine the exact type of stenosis present.

*Brock s[23] Technic* —A left inframammary curved incision is made through the left fifth intercostal space with detachment of the pectoral muscles at their origin The pericardium is opened Some inject 6 ml of 4 per cent procaine solution into the pericardial sac and leave it there for five minutes The pericardium is incised in front of the phrenic nerve Intravenous *procaine helps reduce cardiac irritability Tachycardia is a dangerous* sign and the anesthesiologist must insure adequate aeration If the blood pressure falls and the heart shows signs of distress the surgeon should proceed with his valvulotomy It has been shown by experience that if such cardiac changes occur they will not right themselves Delay for cardiac recovery is unwise and may be fatal In his earlier reports Brock used interlocking mattress sutures for hemorrhage control These have been discontinued as unnecessary and even dangerous if the assistant exerts enough pressure to pull them through Two stay sutures are placed at either side of the incision site but are left slack and are present only to alter the position of the heart if necessary Hemorrhage is controlled by digital pressure[24]

An incision is made into but not through the ventricle The entrance into the heart is made by a special Brock probe[24] This probe confirms the valvular obstruction and passes through into the pulmonary artery A special curved valvulotome is now inserted This valvulotome has a slightly curved shaft with a blade like a spearhead and a blunt end The edges next to the probe end are sharp while the ends nearest the surgeon are blunt. This valvulotome is pressed through the opening until it can be felt in the pulmonary artery by the other hand The opening is then dilated by sounds and clamps (See Fig 12 )

Potts devised an ingenious dilator with ribs which expand as they are enlarged by a screw in the handle similar to a double-edged razor [106] This dilator is a valuable adjunct In this operation the stenosed valve is cut transversely which leaves two cusps and permits the valve to be competent It is expected that this cut endothelializes rapidly No excision is performed, as such might produce an insufficiency The follow-up on patients has not been sufficiently long to determine if the valve ever recloses or if any insufficiency develops Likewise, there is no certainty that the enlargement grows with the patient and is large enough with older ages While 9 of Brock's 43 patients died, only 3 died who were not in heart failure when operated

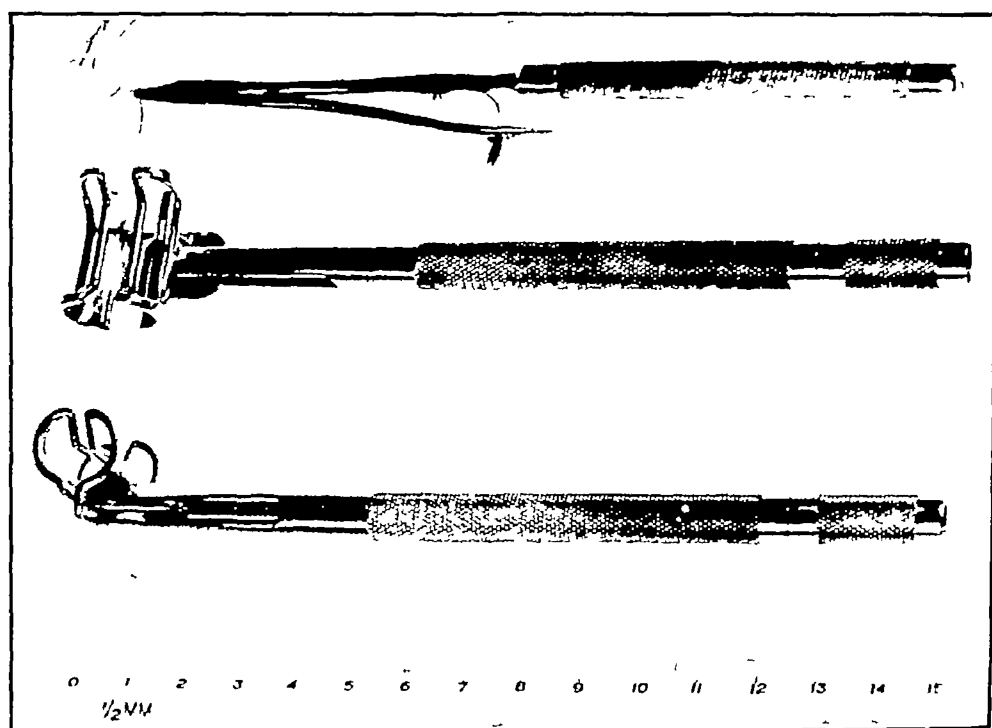



FIG 11 —Potts' clamps and needle holder Clamp occludes portion of vessel for anastomosing while permitting circulation in the rest of the vessel Needle holder holds minute needle and permits its introduction in deep wound. (Courtesy Dr W. Potts, Chicago )

*Valvulotomy for Tetralogy of Fallot* —Brock uses this same operation in over one-half of the patients he sees who have Fallot's tetralogy (22 of his last 50 patients) [25a] He believes that it is important to relieve the obstructive process rather than to by-pass it as in the recirculating procedures Admittedly, it is too early to evaluate correctly the two procedures

Blalock favors the by-pass operation as does Potts [23] Bailey [9] uses the Brock procedure even in tetralogy patients, as does Humphreys [63] The selection of patients, as well as the selection of operations, thus, finally has not been determined It appears that valvular stenosis occurs in a high percentage of patients who have tetralogy of Fallot (40 to 50 per cent, Brock, 40 per cent, Keith, "about half," Sellors and Belcher )[20, 69, 111]

*Infundibular Stenosis* —Brock believes that a direct attack is indicated likewise on the infundibular obstructions He cuts the obstruction ring or diaphragm with a cutting punch

*Special Considerations for the Operation — Anesthesia* —*In no other surgery is anesthesia so important* The less amount of anesthetic agent used the safer it is for the patient The controlled respiration technic gives a quiet operative field The combination of ether and cyclopropane with oxygen during the induction can be followed with cyclopropane and oxygen alone Morphine and atropine or scopolamine removes the anxiety and provides a smooth induction

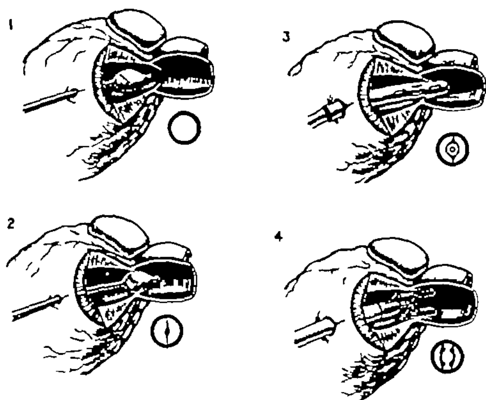



Fig 12 —Brock Procedure Direct valvulotomy for pulmonary stenosis. Valvulotome introduced through the right ventricle between stay sutures. Instruments guided through tricuspid valve by finger pressing against pulmonary artery This permits direct valvulotomy (Courtesy Mr R. C Brock London Eng )

*Hypothermia* is an aid to anesthesia. It can be induced by the use of an ice blanket. The latter is actually a rubber mattress with an inlet and outlet flow for the circulation of a cooling liquid *i e* ice water and alcohol The anoxia, which has been so frequently a complication in operation on the congenital heart patients can be reduced

*Cardiac Arrest* —The main causes for cardiac arrest are inadequate oxygenation or excessive anesthesia It is true that manipulation of the heart *per se* can cause arrhythmia but these variations can be controlled by cessation of such manipulation when such irregularities are noted The anesthesia problem so emphasized by Potts McQuiston[18] and Collins[31] probably causes most of the cardiac arrests The heart stops but it is

secondary to a choking or poisoning by the anesthetic agent in most instances (see Anesthesia, p 27, and Cardiac Arrest, p 45)

## PATENT DUCTUS ARTERIOSUS

The first successful correction of a congenital cardiac vascular defect was performed by Gross in 1938[72] when he successfully ligated a patent ductus arteriosus

Patent ductus arteriosus is a shunt between the aorta and the pulmonary artery This connection, which is essential to fetal life, normally is closed at birth If such a shunt persists, so much blood is diverted from the peripheral circulation to the pulmonary circulation that cardiac hypertrophy follows and heart failure soon develops The occasional patient may live a long and useful span of life In most, however, symptoms occur early Even if children are not incapacitated, serious complications may occur in later life The depletion of the peripheral circulation retards the patient's normal growth These patients are sickly, underdeveloped, and lack the natural defenses of the normal child Infection develops frequently, particularly of the *Streptococcus viridans* type

**Symptoms.**—The symptoms of patent ductus arteriosus vary with the size of the ductus In a few, symptoms are absent, but this depends upon the shunt being a small one and there being no attendant infection In others, the symptoms develop in later life The average age to which these patients live is twenty-four years [12] So much blood may be diverted from the aorta that the individual's physical development is retarded Hypertrophy of the heart with circulatory failure is usual Embolisms from thrombosis in the ductus have been reported Endocarditis or endarteritis of the pulmonary artery may result from superimposition of infection, particularly the *Streptococcus viridans* infection (25 per cent) [1] More rare complications are aneurysmal dilatation or rupture Usually the child will be retarded in growth and weight The characteristic murmur is heard in the second or third intercostal space to the left of the sternum It is a rumbling, continuous murmur, loudest during systole and poorly heard during diastole It has been described as a "machinery" murmur similar to that heard in a work room [62] It is accompanied by a thrill, felt best in systole and in the pulmonary area Since the murmur is present in 98 per cent of the patients, the diagnosis should not be made unless it is heard [44] The murmur may be absent in cardiac failure It is often not heard in infancy. A similar murmur may be heard in patients with septal defects, but such patients have a low diastolic pressure. A normal blood pressure in the femoral artery rules out coarctation

**Diagnosis**—The diagnosis of patent ductus arteriosus is usually not difficult except early in life or when there is a cardiac failure There may be *retarded physical development* (50 per cent) [95] *Dyspnea* depends upon the size of the patent ductus There is a *murmur* heard in the second left intercostal space The murmur is best heard in systole, reduced in diastole but continuous in nature A large patent ductus may occur without a murmur Dammann[39] reported twenty-four patients without a continuous murmur in which the diagnosis was suspected and proven at operation in

fifteen Of the other nine 2 had aortic septal defects and 7 ventricular septal defects, or hearts of the Eisenmenger type All of these patients had pulmonary hypertension and this may account for the absence of the murmur Cardiac catheterization or angiography may help differentiate between an aortic septal defect and patent ductus These tests confirm clinical suspicions [10 11 12] There may or may not be a thrill present If the shunt is large the diastolic blood pressure will be low The electrocardiograph tracing is not typical enough to be of diagnostic value It may be entirely normal or have a *left axis deviation (90 per cent)* [9,10] The roentgen ray, in the presence of a large defect will show a moderate *heart enlargement* particularly of the left side of the heart with fullness of the pulmonary

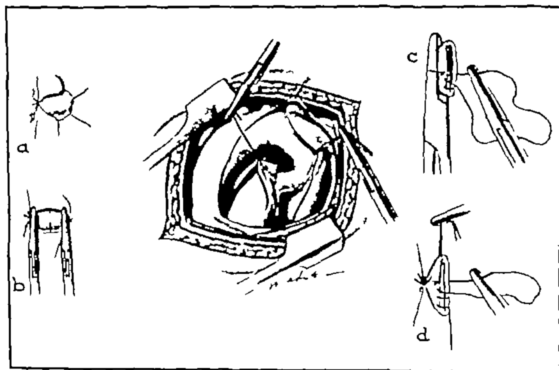



Fig. 13 —Operation for patent ductus arteriosus Incision through 3rd or 4th left interspace Aorta and pulmonary artery dissected free Recurrent laryngeal nerve is guide to ductus. Fat and pericardial reflection removed A Ligature placed prior to clamping B Clamps placed beyond or on ligatures. Ductus divided. C D Suture of the ductus is with continuous 5-0 silk

conus [12] The *heart* on fluoroscopy is *abnormally active* and the pulmonary artery will show a greater amplitude than normal The *hilar dance* of the lung roots is due to an increased vascularity throughout the lung fields from the extra blood supply

*Angiocardiography* —There is an abnormal dilatation of the descending aorta This is just below the isthmus on the anterior aspect Direct visualization of the ductus is unusual [13 14 117 120] Cardiac catheterization usually is not necessary It can aid in measurements of blood flow and is an aid in determining the size of the shunt

**Prognosis.**—Those patients who are alive at seventeen years of age have only one half the life expectancy of the general population [12] Touroff and Vessell showed that the infection accompanying a patent ductus could be cured by ligation of the ductus alone [91 125] This was corroborated by Gross with nine recoveries in 12 infected cases even in the pre-penicillin days [63] The associated subacute bacterial endocarditis, though curable by operation alone, is more safely handled by antibiotic therapy until the infection is eliminated Thereafter, the ductus may be closed [83]

**Treatment.**—Surgical treatment is advisable because of the poor prognosis for life [21]

*Operative Technic —Incision* —Gross suggested an incision on the left side of the chest just under the breast through the third interspace [70] The costal cartilages can be divided to enlarge the wound Others prefer a fourth lateral interspace for children and a fifth rib incision for adults [8] The pleura is opened The fatty tissue around the aorta and pulmonary artery form somewhat of a triangle at the apex of which is the ductus. The pericardial sac may cover part of the dissection It should be freed and, if necessary, it may be opened to aid in identification of the structures The aorta is isolated first above and below the ductus so that it could be controlled should a catastrophe occur The recurrent laryngeal nerve is the guide to the ductus site The ductus is identified and dissected free This is technically difficult, especially in adults, due to the increased vascularity, adhesions, and lack of resiliency of the large vessels The great danger is on the pulmonary artery side The tissues are thin and the pulmonary vessel is difficult to dissect free from its surrounding fat Most of the accidents have occurred from hemorrhage during the dissection of the pulmonary side The fat tissue and the pericardium must be completely reflected before any ligation is attempted Its presence will interfere with ligation and clamps may slip.

The manner of obliterating the ductus has caused some difference of opinion Gross now performs a complete obliteration of the ductus by ligating and dividing it [76,72] Simple ligations in continuity were followed by a re-establishment of the ductus in approximately 10 to 20 per cent of his cases In his hands, there has been no mortality from hemorrhage in 369 divisions of the ductus No other series of patients is comparable to this one His mortality of 2 1 per cent is a striking tribute to the technical ability of his clinic

Scott reported on 180 patients who were operated at the Johns Hopkins Hospital from 1942 to 1948 [113] Their technic includes ligation with multiple sutures This suture ligation technic has been successful and there has been no recurrence in 161 patients Jones uses either method, depending upon the length of the ductus [86] Freeman places a modified Potts clamp before applying his ligature [48] Wangensteen follows Gross's method [126] In general, it can be stated that the measure employed most safely will depend upon the surgeon's skill To inform a surgeon who ligates an occasional patent ductus that he must divide it in order to be successful will result in serious accidents In addition, despite others' reports, occasionally a ductus will be encountered which is too short to be ligated and divided safely In such instances, the technic of Blalock with the ligation and

transfixion technic will be successful and safe Where the communication is of average size ligation division and suture is the procedure of choice For safety in the treatment of the ductus a silk suture can be placed on the aortic and pulmonary sides with ligation in continuity If there is sufficient length a second suture or clamps can be applied, the ductus divided and the ends sutured (see Fig 13) This preliminary ligation in continuity prevents the catastrophe which follows a sprung or slipped clamp The thinness of the vessel wall on the pulmonary side must be considered both in ligation and application and in the handling of any clamp The accidents occur from a tear into the pulmonary side of the anastomosis (see Fig 13) Should hemorrhage occur it is best controlled with the operator's hand No effort to correct such an emergency should be made until arterial transfusions have restored the pulse and blood pressure to near normal any panic has subsided and the program thereafter has been planned and explained to the assistants Gross[186] told of holding such a lacerated pulmonary vessel until 5000 cc of blood had been supplied to the patient and then suturing the laceration successfully in a patient restored to a near normal rather than moribund status

## COARCTATION OF THE AORTA

**Definition and Etiology** —Coarctation of the aorta is a narrowing or a nearly complete occlusion of the aorta The constriction is of the hour glass type Two forms of this aortic obliteration are recognized In the first one there is such diffuse narrowing of the distal portion of the aorta that death occurs very early in life This is called the *infantile type* The second form the so-called *adult type* is one in which there is less severe coarctation and sufficient blood passes the block for the patient to live The first report on the coarctation of the aorta was made by Paris in 1791[191] That the condition is not too rare was shown by Abbott when she collected 200 cases proven by autopsies in 1928[3] Of 1,000 patients with congenital cardiac disease in her series 85 had primary coarctation and it was a complication in 93 others It occurs in 0 1 of 1 per cent of the population[192]

**Symptoms.**—The *infantile type* usually causes death from cardiac failure and rarely exists as a single lesion If the patient survives a patent ductus usually is present This lesion usually is in the form of a diffuse narrowing of the isthmus of the aorta between the left subclavian artery and the ductus. In the *adult type* the symptoms of coarctation of the aorta vary with the degree of obstruction and its duration A few patients may go through life with little or no disability The symptoms are due to constriction of the aorta and development of collateral circulation. One of the earliest symptoms will be *hypertension* in the arms due to the block The blood pressures should be taken in all four limbs The femoral pulsations and those more distal will be restricted or absent as will the blood pressure.

Many *collateral vessels* develop if the patient survives The collateral circulation develops in the superior intercostal the inferior thyroid the transverse cervical and scapular and the subscapular arteries all receiving their blood from the ascending aorta To these are added the internal

mammaries, the epigastric arteries and their branches Gross[69] has called attention to these pulsating tortuous collateral vessels which occur above and below the clavicle, in the axilla, on the back, and below and inside the scapula These are significant in the diagnosis. There is a *murmur*, which is usually systolic in type, transmitted to the back With the reduced blood supply, exercise will cause a blanching of the lower extremities and a flushing of the head and neck and upper extremities The electrocardiograph findings are not constant or typical, but there may be a left axis deviation The roentgen ray will show a mild to a moderate enlargement of the heart and the absence of the aortic knob There is a typical enlarging or *scalloping* of the lower borders of the lateral superior parts *of the ribs* caused by the increased pulsation of the intercostal vessels which develop as collateral circulation This sign usually occurs in those over twenty years of age Headache is a common symptom, as are numbness and pains in the legs

Precision in diagnosis and localization of the lesion is possible now with aortography and each patient should be so studied

**Prognosis.**—Forty per cent of the patients with coarctation of the aorta will die between the ages of sixteen and thirty years, 60 to 74 per cent will be dead by the age of 40 [3,35,37,53,54,65,109] In the infantile type, death most often will be due to rupture of the major vessel. Another group may go on until the third or fourth decade of life and die suddenly of a rupture of the aorta Thirty-nine cases of combined coarctation and rupture of the aorta have been reported [2,27,74,75,77,88,90,112,119] This accident is accompanied by some degenerating disease such as arteriosclerosis Many of these patients develop *Streptococcus viridans* endocarditis Those who survive these complications will have hypertension In most instances, death will be due to secondary cardiac failure or cerebral hemorrhage. The mortality after operation has been less than 10 per cent [65]

**Diagnosis**—The diagnosis of coarctation of the aorta is not difficult if the symptoms are kept in mind The important diagnostic points have been outlined under the symptoms On fluoroscopic and $x$-ray examination, the aorta may be enlarged The heart is not enlarged unless failure occurs Notching of the ribs with scalloping below the margins is diagnostic. With the angiocardiograph, the coarctation may be shown by inserting a catheter into the radial artery An important diagnostic point is the recording of the arterial pressures in the upper and lower extremities

**Treatment.**—Gross,[65,66,67,71] Blalock,[21] Crafoord,[36,37] Humphreys,[82,83,84] Glenn[52,53,54] and others have contributed to the present day surgical treatment of this condition The first operations in 1945 were made independently by Crafoord and Gross Over 300 operations have been performed [37,71]

The object of this operation is to resect the stricture area of the aorta and reunite the ends or, if necessary, insert a graft so that at the conclusion of the operation there is a wide aortic channel available for blood passage

**Operative Technic**—The patient is placed on his right side with the left arm extended out of the field. The incision is made over the fourth or fifth rib laterally and posteriorly If necessary, the entire fourth or fifth rib may be resected and further exposure obtained by dividing ad-

jacent ribs near their sternal junction The opening of the chest is time-consuming because of the collateral circulation, all of which must be carefully resected and ligated Sufficient blood supply for transfusion is axiomatic. The pleura is entered and the lung partially collapsed to expose the site of the coarctation The mediastinal pleura is incised to expose the aorta the subclavian artery the ligamentum arteriosum and the enlarged intercostal arteries Enough of the intercostal arteries are ligated and divided to mobilize the aorta above and below the site of the coarctation. The subclavian artery and the aorta above are freed Rubber shod clamps are put in place on the aorta above the subclavian on the subclavian and on the aorta below the coarctation Rubber tapes are placed behind these clamps for control if a clamp slips The coarctation then is excised at a site which will permit a normal-sized aorta to be anastomosed

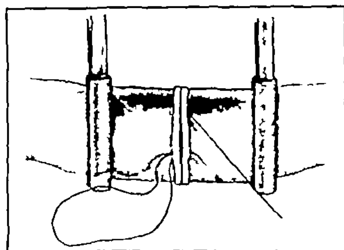


FIG 14.—Repair of coarctation of aorta. Everting mattress sutures inserted In children these should be interrupted to permit growth of vessel with the child

The suturing is then begun using five-O atraumatic silk to approximate intima to intima In the adult a continuous suture may be used If the operation is on a younger individual interrupted sutures are indicated In either case everted mattress sutures are safer and stronger because of the tension following the resection After the anastomosis is complete the patient is placed in a Trendelenberg position The clamp distal to the anastomosis is first removed Any slight bleeding usually can be controlled with pressure The clamp on the subclavian artery is next removed Lastly and slowly the clamp on the aorta proximal to the anastomosis is released Umbilical tapes may be placed above and below the coarctation to help in resection Potts has devised a vice to hold the coarctation clamps This requires division of all the collateral vessels.[18] The clamps are then applied above and below the segment to be resected leaving room for suturing The clamps are then placed in a vice and they can be approximated by a thumb screw Suturing is made simpler by this method A sudden drop in pressure with a raise in pulse rate follows the removal of clamps

Transfusion blood is forced under pressure by the previously placed cut down cannulas This blood helps in correcting the blood pressure drop and pulse rise The parietal pleura is then reapproximated, the lung expanded and the chest wall closed

*Aortic Grafts* —In some patients, it has been impossible to bridge the gap after excision of a coarctation This occurs particularly in the adult type and where there are long areas of narrowing Aortic grafts from other humans have been employed with success The vena cava has been used The subclavian artery has been employed as a graft in coarctation An arterial homograft has been inserted for coarctation 19 times by Gross[61] In the 17 survivors, there has been no aneurysm or rupture of the aorta The results while considered excellent in 14 of the patients did not compare favorably with the end to end anastomosis inasmuch as the grafts tend to shrink In patients where no anastomosis of the ends is possible however, the graft is feasible and lifesaving[13,36,73,115]

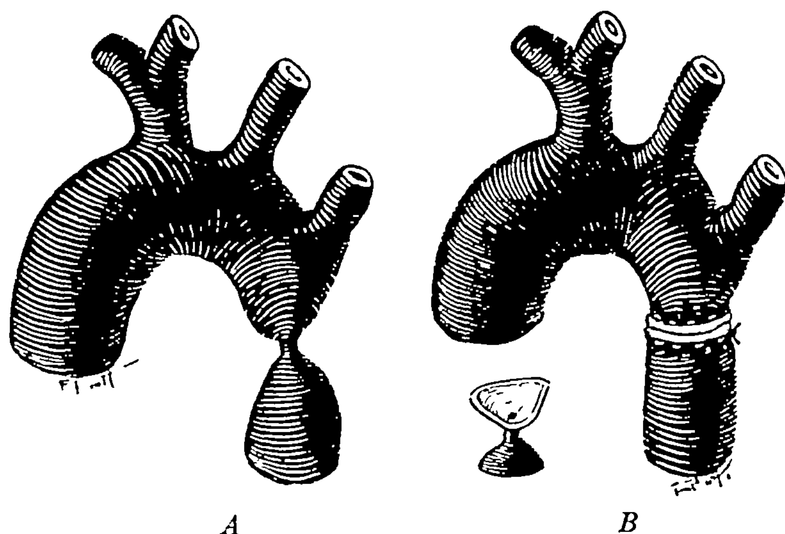



Fig 15 —*A*, Coarction of the aorta treated by excision *B*, Excision and end to end suture Everting mattress-type suture through all of the walls (Gross, courtesy of Surg , Gynec and Obst )

*Coarctation in the Infant* —While the treatment of this lesion in the very young usually has been unsuccessful, the early cardiac failure in the young child may require operative intervention as a lifesaving measure Kirklin *et al* recently reported a successful operation in a ten-week-old infant.[92] This success will alter the lower age range in those patients who obviously will not survive to the age of optimum surgical intervention The mortality will always be higher in the young All four of Burford's[31] infants so treated died The interrupted everting mattress suture is advocated for use in all surgery where the patient is young or where tension on the suture exists.[76]

Gross advised that the operation be performed between the ages of six and eighteen years, when the aorta is more elastic and less sclerotic The aorta can be elongated by mobilization, and defects as long as two cm. have been bridged successfully

Relief of the hypertension in the arms and an increase in the arterial pressure in the legs can be expected There has been no evidence of renal ischemia Gross stressed that there must be age restrictions with respect to performing the operation [71] Below the age of six or seven years the aorta is quite small for the operation and above twenty five years it is too sclerotic It is necessary then that the diagnosis be made early in life

## ANOMALIES OF THE ARCH OF THE AORTA AND ITS BRANCHES

Gross[61] classified anomalies of the aortic arch Autopsies have shown many unusual aortic arch anomalies Their recognition during the life span of the individual has been possible and with better roentgen ray diagnostic technics the number will be increased

A classification of various anomalies taken from Gross and Ware[61] follows

- I Right Aortic Arch
  - A Inversion of the viscera
    Inversion presents no surgical problem
  - B Right aortic arch without viscera inversion
    - 1 Anterior type
      Aorta descends on the right but the aortic arch is in front of the trachea
    - 2 Posterior type
      Aorta descends to the right but arches pass to the left *behind* the esophagus Three such groups are recognized
      - (a) Right aortic arch with left subclavian artery arising from the arch crossing *behind* the esophagus
      - (b) Right aortic arch with no blood vessel crossing the mid line *behind* the esophagus (A vessel may pass in front of the trachea )
      - (c) Right aortic arch with the left subclavian artery and ligamentum arteriosum arising from a persistent left aortic diverticulum
- II Double Aortic Arch with or without one of the limbs obliterated
- III Right Subclavian Artery arising from the *left side* of a Normal Aortic Arch and crossing the Midline Compressing the Esophagus
- IV Patent Ductus Arteriosus
- V Coarctation of the Aorta

**Right Aortic Arch.**—This is a persistence of the embryonic right fourth brachial artery to form the aorta instead of the usual left fourth one The posterior type (arch behind the esophagus) is more common than the anterior one (arch in front of the trachea) The posterior type causes compression of the esophagus The left carotid artery usually arises on the right side and tends to form pressure and compression of the trachea

SYMPTOMS —The right aortic arch anomalies may cause no symptoms There may be dyspnea cough pain dysphagia and occasionally dysphonia due to pressure on these parts

SURGICAL TREATMENT —Surgical correction of many of these arch anomalies is now possible Where a smaller vessel such as the carotid or

subclavian artery, causes the pressure or obstruction, it can be ligated, transfixed, and divided. The vessel may be displaced and sutured to some contiguous structure if necessary to maintain the displacement after its division.

**Double Aortic Arch.**—In some of the lower forms of animal life, a double aorta is usual. In man, if both the right and left fourth brachial arches persist, a bifid aorta will result. In most of these, one limb becomes obliterated. These two limbs may surround any mediastinal structure, but in most of the cases, the esophagus or trachea, or both, are so surrounded.

SYMPTOMS.—There may be no symptoms. *A patient living to an old* age was described by Curnow.[38] If the two limbs of the arch are at all close together, however, there will be compression and stricture of some viscera and structures. Most of these patients have sufficient difficulty to come to the attention of the physician in infancy. Early symptoms depend on what organs are compressed. The usual signs are stridor and dysphagia, beginning soon after birth. Severe coughing is present with the dyspnea. There may be a noisy, wheezing cry. There is difficulty in swallowing which is made worse by the rapid breathing necessary for the child to aerate the lungs.

SURGICAL TREATMENT.—Of the sixteen patients coming to operation by Gross, 13 had a left descending aorta. The mortality rate of 25 per cent makes it necessary to reserve the operation for those with severe compression symptoms.[61] The treatment depends on which limb is the larger and which one anatomically should persist to carry the blood. The decision as to which limb to divide will depend upon which one carries the greatest blood supply. In some, only a vestigial remnant of the arch will be present. Such a band may cause more severe symptoms, because by its narrowness it causes more pressure and constriction. Division of this congenital remnant may relieve all the symptoms.

**Anomalous Right Subclavian Artery.**—This is a more frequent anomaly and appears when the right subclavian artery arises from the left side of the arch. To reach the right side and its normal point of exit from the chest, it must cross from the left to the right side of the thorax. It may thus run behind the esophagus (in 107 of Holzapfel's[70] cases), between the esophagus and trachea (in 20 of the same series), *or in front of the trachea* (in 6 of the same series). An anomalous right subclavian artery occurs in from 0.4 to 1.6 per cent autopsies. The author has operated on 2 patients with congenital arteriovenous aneurysms in which the right subclavian artery arose on the left side.

SYMPTOMS.—In some patients with an anomalous right subclavian artery there may be no symptoms. Many are found only on postmortem examinations. The most common symptom is difficulty in swallowing. This may occur early in life, or may be delayed until the blood vessel increases in size or becomes sclerosed. Respiratory symptoms do not occur.[61]

DIAGNOSIS. *The diagnosis is made on the symptoms of dysphagia and* confirmed by roentgen ray examination using a contrast dye. Fluoroscopic evidence of difficulty in swallowing the dye is of diagnostic importance.

SURGICAL TREATMENT — As the anomaly involves the first portion of the subclavian artery resection of the artery can be done with impunity because of the excellent collateral circulation which develops With improved roentgen ray technics more of these patients will be identified and the operation will be performed more frequently Many patients now treated for esophageal spasm or constriction may be in this group Some patients now under psychiatric care for so-called functional dysphagia may fit well in this group All of these patients should be reexamined to see if there is an organic rather than a mental reason for their complaints and difficulty

In the treatment of the compression of the trachea or esophagus by vascular anomalies certain technical points have been emphasized The attendant pulmonary infection should be treated A cut-down polythene tube should be available for rapid transfusion An intratracheal tube made of a soft material and a closed anesthesia system is imperative Since most of the anomalies are on the left side the approach should be through the left anterolateral chest and pleura The importance of attendant fibrous bands as a cause for the constriction should be remembered and these must be divided The postoperative management as in any intrathoracic operation requires skilled and continuous observation to prevent the accidents which determine the success or failure of chest procedures

## OTHER CARDIAC ANOMALIES

**Aortic Septal Defects.**—This condition results from a congenital communication between the aorta and pulmonary artery just above the aortic and pulmonary valves This gives a shunt from the left to the right side of the heart The physical findings are similar to those of patent ductus arteriosus The two conditions can be differentiated at times by aortograms.[illegible] Cardiac hypertrophy and failure may result from the strain The surgical repair of such a defect has been successfully performed by Gross[illegible] In his case report the opening between the two vessels was closed by a piece of 1 cm linen tape The possibility of successful closure of such defects thus exists

**Abnormalities of the Pulmonary Vessels** —Anomalous drainage of the pulmonary veins into the right side of the heart has been reported in 154 instances[illegible, 47, illegible] These and other anomalies will be encountered from time to time with the relative frequency with which the chest is opened surgically The diagnosis is confirmed by angiocardiography and by finding that the oxygen content of the blood of the right side of the heart is about the same as in a peripheral artery These lesions have not been classified nor are therapeutic surgical procedures for them completed Their surgical importance during pneumonectomy or lobectomy may be of concern in pulmonary resection The identification of the blood supply to and from the remaining lung must be ascertained to prevent accidents such as shutting off the blood supply to or from the remaining lung

**Transposition of the Aorta and Pulmonary Artery** —The two great vessels may arise from reverse ventricles the aorta from the right and the pulmonary artery from the left chamber this condition being called a complete transposition In the corrected transposition these vessels arise from their normal ventricle but in reversed relationship[illegible]

In the complete transposition blood goes from the left ventricle through the pulmonary artery to the lungs and returns by the pulmonary veins to the left auricle and to the left ventricle. Reversely, blood from the right ventricle goes to the aorta and is returned by the systemic veins to the right auricle and right ventricle. The two systems are separate.[22] Life is incompatible with such a circulation, but some patients live due to septal defects. Of the 123 patients reported, the life expanse was five and a half months, although 6 lived ten years or longer.[76] An interventricular septal defect is the compensatory mechanism. Of the operations performed, an extracardiac venous or arterial shunt, the creation of an auricular septal defect, or a combination of both were tried. Twenty-two of the 28 patients operated died. More recently, an auricular septal defect or an anastomosis between the right subclavian and pulmonary artery has been created. Eight of 12 patients so operated have lived.[32] A report of a complete transposition of the main arterial stems recently was made.[100] Usually this status is incompatible with more than transient survival. This patient lived twenty-nine years. Compensation occurred by atrial and ventricular septal defects. This patient also had a patent ductus arteriosus and a coarctation of the aorta. She was cyanotic and limited in exercise tolerance but was in heart failure only in her last three years of life. No surgical therapy for such a condition has been evolved.

**Congenital Interatrial Communications and Defects.**—ETIOLOGY.—This defect exists more frequently than was supposed. It may occur with other defects. A few patients live a normal span of life. Most do not. The lesion develops due to a disruption of the normal developmental process of the separation of the two atrial cavities. The most common interatrial communication is a *patent foramen ovale*. This is due to failure of the primary and secondary septums to fuse. The size of such foramen varies from a pinhead to one that admits a finger. If the primary septum fails to fuse with the endocardial cushions there may be a defect just above the mitral and tricuspid valves (*persistent ostium primum*). If the lower portion of the septum primum and of the membranous part of the interventricular septum results in *atrioventricularis communis* then all four chambers are connected. If the secondary septum is absent a defect develops in the upper portion of the primary septum, the *foramen secundum*. The *atrial septum* also may be completely *absent*.

In the usual defects the shunt is from left to right. The right ventricle has an increased filling, as has the pulmonary flow. The right ventricle hypertrophies and the pulmonary arterial tree dilates. The right atrium must enlarge to receive the extra blood flow. The left heart remains the same. The left atrium, although receiving larger amounts of blood, does not dilate as it shunts the blood through the defect. The left ventricle receives little of the blood and does not enlarge. The hypoplasia of the aorta described before is likely relative to the increased size of the pulmonary artery since the aorta carries the same amount of blood with the usual compensation mechanism. This defect occurs in from 2 to 24 per cent of congenital cardiac lesions as the sole lesion and in 33 to 40 per cent as the only lesion or in combination with other lesions.[1] The condition is more prevalent in the female.[110]

SYMPTOMS.—These patients develop poorly. They are weak, pale with translucent skin. Venous pulsations are frequently seen. The heart is enlarged and a systolic thrill is felt in the second interspace. The murmur is atypical. Rheumatic disease causes mitral murmurs. The systolic murmur at the base and a systolic or biphasic bruit at the apex are considered typical of the atrial septic defect with mitral stenosis (Lutembacher's syndrome).[illegible] Cyanosis is present, usually only in the patients who develop failure and disappears with compensation. Continuous cyanosis in the absence of failure means a large septal defect is present or a pulmonary stenosis also exists with a blood shunt from the right to left side. Pulmonary vessel resistance may cause the right to left flow. Pneumonia and right heart failure to which these patients are susceptible cause cyanosis. Clubbing occurs after a time. Dyspnea is a frequent sign. Limited exercise ability, tachycardia, chest pain and rheumatic heart signs are frequent. The *prognosis* varies greatly. It is better than many of the other congenital malformations but the signs accompanying it make life not too satisfactory for the majority. The diagnosis is made on the symptoms and signs and by ruling out other lesions. It can be confirmed by special tests. Angiocardiographic studies may be diagnostic but are dangerous. Cardiac catheterization is diagnostic. The catheter can be passed through the defect if introduced by way of the inferior vena cava. Pressure readings and oxygen determinations will demonstrate the shunt and whether there is a coexisting pulmonary stenosis.

SURGICAL TREATMENT.—The surgical treatment was developed by animal experimentation and is far from satisfactory. It is mentioned to demonstrate the surgical future of cardiac disease. Steps are being taken to eradicate or improve all types of heart trouble and while still faltering show a surgical trend.

METHODS.—Dodrill used a large clamp to press the atrial walls against the septum with exclusion of the rest of the atria.[illegible] This produced a dry field for suturing the defect. In some cases the defect was sewed to the atrial wall. Swan[illegible] devised a method of invaginating the two atrial appendages. Silk sutures are passed through the appendages by way of a probe and these are sutured over a circular polythene button on either side protected by gel-foam. When these buttons are snugged tight the two appendages are drawn against the defect. Hufnagel[illegible] developed a rod clamp which when turned into another rod permits nylon buttons to be drawn against each other. One of these buttons is supplied with teeth. This works quite satisfactorily in animals. Cohn[34] closed defects in dogs by pulling the atrial wall as a patch to close the defect. A wire encircled a part of the wall. Thereafter, silk sutures were used to bury the portion closing the defect. Murray[illegible] pulled silk sutures through the atrium, the sutures being introduced to the right of the aorta and the pulmonary artery and posteriorly through the area between the superior vena cava and the right pulmonary veins. These sutures were tied posteriorly and tightened anteriorly.[illegible]

The difficulties of such technics included thrombus formation, difficulty of closing the defect by suture, the addition of foreign bodies and the danger of interference with atrial flow. Bailey's[16] procedure took advantage of the fact that in the septal defects there is a dilated right atrium or excess

of tissue while at the defect there is a lack of tissue Using the intracardiac finger as a guide, sutures were placed through the right atrial wall, through the defect and again out through the atrial wall approximating this wall to the marginal defect This closed the communication between the two atria The right auricular appendage which had been used during the intracardiac part of the operation then communicated with the left auricle This appendage was then oversewn Bailey gave the name of atrio-septo-pexy to this procedure While not answering all the questions raised by this problem, it is a step forward

Swan[121] more recently has varied his technic He now opens the right auricular appendage, inserts a polyethylene button and passes the sutures through this button out through the other auricular appendage This button was larger than the defect as determined by the intracardiac finger The sutures through the left appendage then are tied over a second button This tying invaginates the left appendage and the button is firmly pressed against the defect This method has the advantage of a guided placement and the button is anchored to tissue The difficulty of thrombus or slough remains but his experimental work and limited clinical experience appears to indicate this method may be applicable in some defects

Gross recently has employed an open type of operation [61] He sews a rubber well over the auricle and opens it He reports no difficulty in making the well attachment liquid-tight The blood enters the well to the height of the auricular pressure (from 5 to 10 cm ) The blood is kept fluid with heparin The defects are then closed by working through the blood If the defects are small they can be sutured Where they are larger, they are closed with a polythene covering sutured around the defect This method has worked well on human patients

The difficulties of this type of repair are many The foreign buttons, etc , which work so well on dogs are not satisfactory in humans where the edges of the defect are small, thin and do not hold sutures like the normal dog heart wall Again the advent of a sure extracardiac circulation apparatus is awaited to complete surgical technics adequately and to add to those devised under difficult circumstances.

**Interventricular Septal Defects.**—Ventricular septal defects are caused by embryological failures with the exception[111, 123] of the occasional rupture due to muscle necrosis, or the trauma of a stab or gunshot wound These defects are the low or the high type [5] Since the pressure in the two ventricles is equal and usually zero except during cardiac systole, there is little interventricular flow This is not like the auricular defects where the pressure change causes a constant loss of blood. If the low defect is very large, the heart acts as it would with a single ventricle, pumping oxygenated and unoxygenated blood into both the systemic and pulmonary systems The high ventricular defects are larger and the physiologic change in the individual is greater Many of these defects are associated with an infundibular pulmonary stenosis and an overriding of the aorta. Small defects have been corrected at the time of the infundibular pulmonary stenosis operation A cone-like section of the pericardium is rolled up and sewn and this is pulled through the defect by a suture, much as is a cork [11] This can be done through the right ventricle at the time the operation of Brock is performed (See page 80 )

In the large defects efforts to apply a patch of pericardium or muscle wall have not succeeded. These patches can be attached to the wall by sutures pulled through with a probe, but animal experiments show that they do not remain attached to the ventricle wall. The possibility that some other part of the body can be used for closure exists. Skin offers such a hope. Once the extracardiac circulation problem for operation has been settled it is possible that a section of the hypertrophied heart muscle may be used effectively.

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# Chapter 10

# SURGICAL TREATMENT OF ACQUIRED CARDIAC VALVULAR DISEASE AND ITS COMPLICATIONS

*Mitral Stenosis; Mitral Insufficiency; Aortic Stenosis, Mitral and Aortic Stenosis, Tricuspid Stenosis; Congestive Heart Failure*

ACQUIRED heart disease is the greatest single cause of deaths today When we combine this disease with its allied and complicating changes in the blood vessels, kidneys and brain, we account for 3 out of 4 deaths of those surviving infancy The school-aged child, particularly, is susceptible. Approximately one-sixth of our population (26,000,000) are enrolled in elementary and high schools in the country in any one month At least one-fourth of the deaths from five to nineteen years of age result from heart disease [66]

The most common heart lesion is mitral stenosis, which afflicts 85 per cent of the million rheumatic heart patients in the United States [35] In areas where rheumatic fever is endemic, this figure rises [67]

Most such lesions result in death by middle age The aortic valve also may be stenosed by rheumatic fever and in some of the luetic lesions partial obstruction of this valve also occurs [11]

With the great improvement in anesthesia, antibiotic therapy, and blood replacement, surgical efforts previously considered impossible can now be carried out successfully Since the stenotic lesion is a mechanical one (pure mitral stenosis occurs in 10 per cent[69]), its relief in many patients can be accomplished mechanically Even where an insufficiency of the valve develops concurrently, relief can be obtained in many of these patients This subject will be discussed under mitral, aortic and tricuspid acquired lesions

**Historical.**—Since Rehn successfully sutured a heart in 1897,[59] cardiac surgery has been a challenge to all surgeons The first to suggest that mitral stenosis could be surgically corrected was Sir Lauder Brunton [18]

In 1914, Tuffier attempted to relieve an aortic stenosis. He opened the chest above the aorta with the intention of passing a knife through the aorta and cutting the stenosed valve The technic was changed on the operating table and he merely dilated the valve by invaginating the aortic wall on his finger This patient lived as long as 1924 (10 years) and was the first successful result reported [62, 64]

The first surgical attempt to relieve a stenosed valve was by Doyen in 1913 His operation on a congenital pulmonary stenosis failed [30] No further reports of surgery of this type appeared until Allen and Graham's attempt to relieve mitral stenosis with the cardioscope in 1922 [1] In 1924,

Cutler and his associates began their experimental work.[24] Souttar[41] in England, Pribram[44] in Germany and Powers and his associates[46] had been working on the problem experimentally. The mortality in 12 patients operated up to 1928 was very high: 9 deaths of 12 patients—one on the operating table and 8 a few hours or a few days after operation.[40] It was of interest that of the 3 who recovered improvement was reported and 2 of these had been treated by digital dilatation alone.

RESULTS OF 12 EARLY OPERATIONS

| *Method* | *Patients* | *Deaths* |
|---|---|---|
| Tenotome knife | 4 | 4 |
| Blind partial excision with cardiovalvulotome | 5 | 4 |
| Digital dilatation | 2 | 0 |
| Partial excision of scarred valve by direct vision with the cardioscope | 1 | 1 |
| | — | — |
| | 12 | 9 (83 per cent) |

Eighteen years went by thereafter before any constructive work in this field could be reported. In 1945 Bailey's original work on animals was begun and this was followed soon by its application to human patients.[3] While the early mortality was so great as to cause the surgical attempts to be discontinued, it was not a greater mortality than that which followed early operations on the stomach. For example, W. W. Keen[47] reported in his Cartwright Lectures that all 28 gastroenterostomies performed up to 1875 had died. The mortality for gastroenterostomy up to 1885 (35 operations) was still 65.7 per cent. In 1884 the mortality rate for gastrectomy was still 81.6 per cent. Thus it is apparent that even in this surgically advanced age any new surgical attack on a part of the body previously not operated will have a high mortality until the surgeon's experience and technic and selection of patients have overcome the early and unavoidable errors. It is a regrettable fact that the dictum of Deaver—that a surgeon's success comes only after he has filled a graveyard—is true. Such early deaths, while unfortunate, are sacrificial to the common good, just as were the 120,000 casualties following the atomic explosion at Hiroshima. This blood bath saved hundreds of thousands or millions of American and Japanese lives which would have been lost had a direct invasion on the Japanese home islands been necessary. This comparison is remote but the underlying similarity remains.

In February of 1950 Smithy, whose untimely death removed a great investigator, could report on 8 cardiac operations on 7 patients with 2 deaths, a mortality of 28.6 per cent. Three of the patients had a successful excision of a segment of the mitral stenosed valve by a blind valvulotomy inserted through the ventricle.[60]

The problem of surgery of the heart and particularly stenosed valves has been attacked by two generally different methods. One is the extracardiac approach of which the operation of Blalock[15] for pulmonary stenosis is an example. Gross,[37] Crafoord and Nylin[23a] have also contributed

greatly and brilliantly to this method. A further extracardiac advance has been reported by Bland and Sweet.[32] Their operation to relieve pulmonary edema secondary to advanced mitral stenosis is by performing an extracardiac shunt. They anastomosed the dorsal segment branch of the right inferior pulmonary vein to the azygos vein. This is an extracardiac shunt to reduce the high pressure in the left auricle and pulmonary vein by permitting its escape into the systemic venous bed.[32]

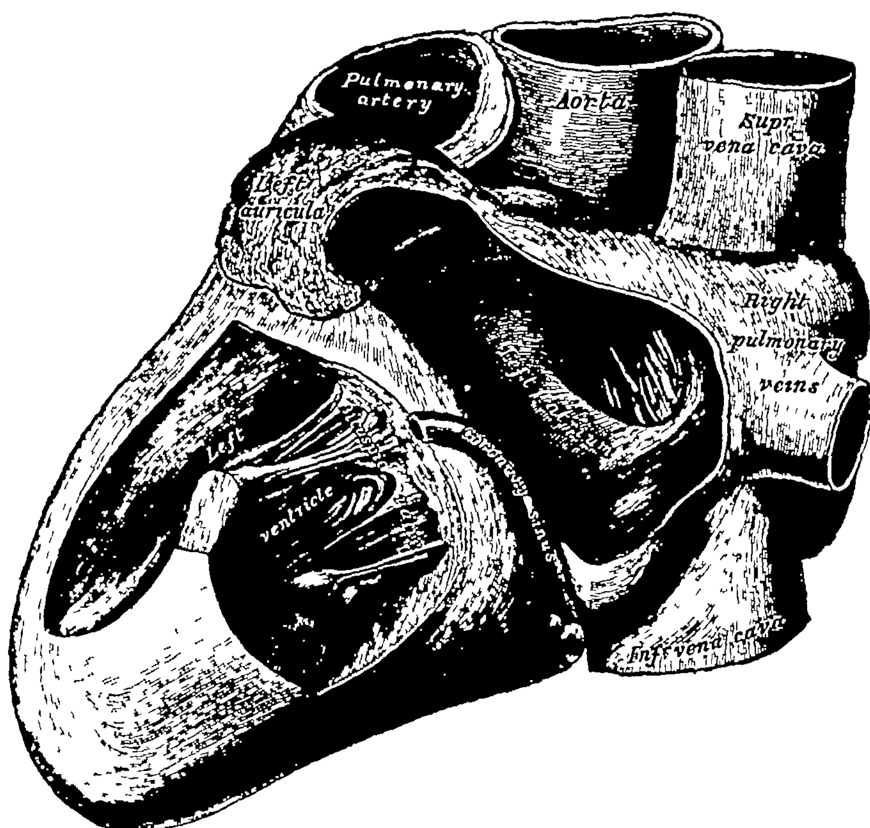



FIG. 16.—Anatomy of the left side of the heart. (Gray's Anatomy.) Auricular appendage usually thicker and fatter. In mitral stenosis the left auricle and pulmonary veins are dilated and occupy a relatively larger part of the heart.

An intracardiac approach has been the dream and aim of most surgeons. When one sees an autopsy on a young, vigorously constructed individual who has died long before his physically appointed time from a mechanical obstruction at the mitral valve, the loss to this closure seems wasteful. The rheumatic heart disease that caused the inflammation in the valves and commissures often has long burned itself out. The scar that healed the inflammation has closed and contracted the commissures. The patient's problem thereafter is a mechanical one. The blood arrives in the left auricle from the pulmonary vein. It cannot get through to the ventricle in sufficient quantity. The blood backs up into the pulmonary veins and lungs with congestive failure and death results. Smithy, Harken and his associates, Brock, Murray, Bailey and Glover, were early workers in the direct approach to the stenotic lesion.[3, 9, 12, 17, 29, 32, 34, 42, 53, 54, 60]

**Anatomy** — The heart is a hollow muscular organ of a somewhat conical form lying between the lungs in the middle mediastinum and enclosed by pericardium. Its position is oblique in the chest behind the sternum and the adjoining parts of the rib cartilages, projecting farther to the left than to the right with two-thirds of it in the left thoracic cavity and one-third of it in the right thoracic cavity. In the adult its average diameter is 12 by 9 cm and it has the thickness of about 6 cm. Its weight varies greatly but in the male it averages from 280 to 340 grams while in the female the variation is from 230 to 280 grams. It tends to increase in weight and size with age, more so in the male than in the female. Septa divides the heart into right and left halves and a constriction further subdivides each cavity into an upper part or atrium and a lower part or ventricle. On the

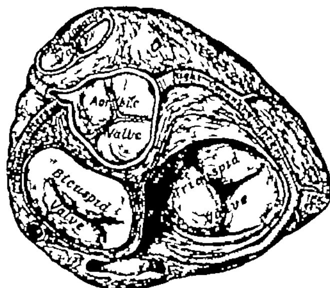



Fig. 17 — Relative position of the valves of the heart. (Gray's Anatomy.) Ventricles exposed by removal of the atria. Position of coronary arteries shows their relative safety during any maneuver through the appendages.

surface the atria are separated from the ventricles by the coronary sulci or grooves which contain the vessels that supply blood to the heart.

The heart is further subdivided into a base and apex. The base points upward, backward and to the right, being separated from the fifth, sixth, seventh and eighth thoracic vertebrae only by the esophagus, aorta and thoracic duct. It is made up mainly of the left atrium and part of the right atrium. The four pulmonary veins, two coming from either side, open into the left atrium while the superior and inferior venae cavae open into the right atrium.

The apex is forward and downward to the left and is covered by the left lung and pleura. Its point is in the fifth left intercostal space approximately 9 cm from the mid-sternal line, which is anatomically 4 cm below and 2 cm medial to the left nipple of the breast.

Since we are particularly interested in the left side of the heart, it is of interest to note that normally the left atrium is smaller than the right but its walls are thicker. This atrium consists of a main cavity and an

auricular appendage The left atrium has the opening of the four pulmonary veins, the orifice from the auricle to the ventricle and the pectinate musculi The left ventricle is longer and more conical in shape than the right It has a nearly circular outline Its walls are three times the thickness of those of the right ventricle In addition to the opening from the left atrium, this left ventricle contains the aortic outlet, the bicuspid or mitral valves, the aortic valves, the trabeculae carneae and the chordae tendineae

MITRAL VALVES —The mitral valve is a bicuspid valve and it is attached to the surrounding of the opening between the auricle and the ventricle. The valve is formed by two triangular cusps which are made by duplicating the lining membrane, this lining membrane being strengthened by fibrous tissue which contains a few muscular fibers. The cusps of this valve are unequal in size, and are longer, thicker, and stronger than those of the tricuspid valve The larger of the two cusps is placed in front and to the right between the ventricular and aortic orifices, and this cusp is known as the anterior or aortic cusp because of its physical location The smaller cusp of this bicuspid valve is also called the posterior cusp and it is placed behind and to the left of this opening In addition, there are two smaller cusps found at the angles or junction of the larger cusps These cusps of the bicuspid valve are attached by chordae tendineae These chordae tendineae arise from the apices and margins of the cusps on the ventricular side and are delicate but tendinous chords These are thicker and stronger but less in number than on the right side of the heart These chordae tendineae arise from papillary muscle and are connected to both cusps of the bicuspid valve [37] The muscle fibers in the trabeculae carneae contribute to the force and energy of the heart as a pump The heart is not a perfect sphere and contracts in a wringing type of motion [20] The papillary muscles have a dual function In addition to helping maintain closure of the atrioventricular leaflets, they assist in emptying the ventricle The muscle fibers of the heart muscle can shorten themselves only about 20 per cent [13,35] These muscles alone can reduce the cardiac diameter thus about 20 per cent and this would empty only 50 per cent of the diastolic blood volume These papillary muscles pull the walls of the ventricle to which they are attached to the center of the ventricle cavity [20] Their action completes ventricular emptying During ventricular systole, the papillary muscles contract at the same time thus drawing the chordae tendineae taut This prevents these valve leaflets from becoming inverted or displaced backwards into the auricle

Thus the normal mitral valve is similar to a cone made of a thin, flexible membrane This truncated cone, with its base attached at the opening of the left auricle into the ventricle, has an apex which extends into the ventricle It is this apex and the apical half of this cone which is suspended by numerous chordae tendineae, or "guy wires"[35] These chordae tendineae attached to papillary muscles arise from the ventricular wall near the ventricular apex In two areas, the chordae tendineae are grouped more heavily, and these areas represent the anatomic junction of the two components of the valve, the anteromedial and posterolateral leaves These leaves fold themselves on this line from the anteromedial to the

posterolateral line These areas thus become the so-called ' commissures Normally during auricular systole the posterolateral cusp is well away from the ventricular wall During ventricular systole the ventricular wall may well approximate and support this posterolateral cusp

This is further modified and improvised by rheumatic heart disease which flattens these cusps.

Mitral regurgitation results from a defect or dysfunction of the antero-medial valve cusp A defect in the posterolateral cusp produces only a limited regurgitation With rheumatic heart disease the vegetations which develop are on the line of the closure of the valve As these heal scar tissue forms With repeated infection and healing the apical portion of the valve cone becomes narrowed and scarred If the condition ceases and there is no further inflammation a mild purse string puckering may result In others where only one- to three-fourths of the cone is involved a flexible margin is left along the base In more advanced disease the whole valve is a rigid completely inflexible opening and it frequently is calcified hard and plaque-like with a small fish mouth like slit in it This classical description of mitral stenosis actually is present in only the advanced diseased patients.

With mitral stenosis the apex of the mitral cone is fixed or held in its flattened position and the commissures are thus a pathologic entity This produces a resistance to the passage of blood from the left auricle into the left ventricle As a result of this fixation there is some regurgitation from the ventricle into the auricle during ventricular systole The rheumatic disease may so shorten the valve as to make it impossible for it to be approximated In such cases the mitral insufficiency becomes the prominent disease

**Physiology** —In mitral stenosis the left ventricle cannot be satisfactorily filled and therefore the output to the systemic circulation is inadequate Patients with hearts of this type are unable to put out sufficient amount of blood except under a quiet or resting stage If these patients work they develop signs of fatigue dizziness or unconsciousness. As a result of the fact that blood cannot leave the left auricle the pressure in this chamber increases and is secondarily transmitted to the entire pulmonary vascular system and thus to the right ventricle Clinically this results in a pulmonary hypertension with exertional pulmonary edema (dyspnea) failure of the right side of the heart with peripheral edema fluid in the abdomen and peritoneum The liver enlarges and at times there is a rupture in the pulmonary capillary bed which causes the hemoptysis or heart failure cells.[4]

It has been shown that a mitral valve area of 1 5 sq cm or greater permits a normal function. If the valve area is 2 sq cm the patient has symptoms only on severe exertion When a valve area becomes 1 0 sq cm. or less the valve area is taut and symptoms develop on slight or no exertion At 1 0 sq cm the valve is only 20 per cent of its normal size Thus, in the selection of patients it may be difficult to determine which valves are larger than 1 0 sq cm but there will be little difficulty in selecting those with the so-called tight valves. One added factor is that after the valve admits one finger (1 5 sq cm in cross section) the regurgitation progressively increases.

Harken's and his associates[41,42,43] early work in the treatment of mitral stenosis duplicated earlier operative efforts. His original poor results using various forms of valvulotomes was similar to that of Cutler, Beck, Bailey, Glover, etc. (9 operations with 6 deaths). Such unfortunate results led to extensive investigation of patients who died of mitral stenosis. Autopsied specimens from these patients and also from their own operated patients who died made them conclude they were unable to predict how or where a valvulotome would cut. It was determined further that in most instances simple manipulation of the stenosed valve with the finger could free the scar at the commissures.

Several types of mitral stenosis have been described, but, in broad terms, there are mainly three.

*Type one* is the most common form (3 out of 4 patients). The valve leaflets are fused in a firm fish mouth-like fashion at the anteromedial and posterolateral commissures. The degree of fibrosis or calcification depends upon the length of time the patient has had the disease, the repetition of the attacks, and the patient's reaction to the disease. In this group, manipulation of the valve by the finger alone has been quite satisfactory. In a *second type* of mitral stenosis, the opening is fish mouth-like but its edges are somewhat elastic. Harken reports that 10 per cent of the stenotic valves he has seen fall in this category. This elasticity precludes simple finger fracture as the valve gives with the finger pressure. Pressure of an instrument or cutting may help. In a *third type* the valve is calcified and cannot be manually fractured safely. This type requires cutting out of a piece of the commissure.

**Treatment of Mitral Stenosis.**—Many factors determine the treatment of mitral stenosis. Obviously, the degree of rheumatic fever, the recurrence of the inflammation, status of the myocardium, the patient's age, degree of obstruction, concomitant mitral insufficiency and other valvular defects are all factors. To this must be added the method and completeness of therapy.

Medical Treatment.—Conservative treatment has a two-fold purpose. The prevention of further rheumatic attacks is primary and in most cases today is successful. The removal of foci of infections such as diseased tonsils is indicated. Undue exposure should be prevented. The antibiotic drugs, vaccines, and vitamin therapy with a balanced diet will help. In addition, the strengthening of the muscle can be accomplished to some extent. In an acute phase, morphine rests the heart muscle and reduces the individual's activity and requirements. This may be aided by the addition of oxygen therapy. Certain drugs have an important part to play in improving the muscle tone and action. Foremost in this category are digitalis, strophanthin, Coramine, and the various respiratory stimulating drugs. In cardiac failure there is a back-flow in the cardiopulmonary circulation. This results in peripheral and pulmonary edema. To help correct this phase the diuretic drugs play a part. In the acute phase, the reduction in the circulatory load by phlebotomy is effective. This reduces the amount of blood which the failing heart must pump through the circulatory system. Thus the relation between the pump, the circulating media and the peripheral resistance is maintained.

In general however such medical measures fight a losing battle One failure is circumvented or relieved only to be followed by another one With each such episode the muscle reserve is depleted Further scarring or other attacks then increase the stenosis and the vicious circle becomes more rapid At a certain stage of stenosis the heart can no longer be compensated and failure and death result This stage varies with different individuals ages, and cardiac status Thus at autopsy we may see a mitral valve which admits one index finger in which there has been no symptoms In a second patient subject to repeated heart attacks and dying in failure the valve may admit only the little fingertip Between these two stages there is a sufficient sized valve to permit cardiac function without failure The selection of the patient who should have surgical intervention requires careful study both clinically and physiologically and in certain cases careful chemical and cardiographic studies are necessary

**Selection of Patients for Commissurotomy**—In selecting patients for commissurotomy both indications and contraindications must be considered Some patients may be poor selections both from the indications and contraindications standpoint As our knowledge increases so will our ability develop to advise surgery for the correct patients at the right time The greatest progress must be made in the selection of the patient for the operation at the right time in his disease

SELECTION OF PATIENTS

1 *Most Satisfactory Prognosis*
   (a) Age under fifty
   (b) No active rheumatic heart disease
   (c) Adequate response to exercise test
   (d) Normal sinus rhythm
   (e) Out of cardiac failure
   (f) Lesion purely (10 per cent) or predominately stenosis
   (g) No embolic or cerebral vascular phenomena
2 *Less Satisfactory Prognosis*
   (a) Age under sixty
   (b) More than one attack of heart failure
   (c) Only fair response to exercise test
   (d) Auricular fibrillation with mid-systolic bruit
   (e) Left auricle and pulmonary vessels markedly dilated
   (f) EKG shows myocardial damage (T wave)
3 *Least Satisfactory Prognosis*
   (a) Age over sixty
   (b) Many attacks of heart failure—but controlled
   (c) Poor response to exercise test
   (d) Auricular fibrillation with late systolic bruit
   (e) Mitral insufficiency
   (f) Embolic or cerebral vascular phenomena
4 *Contraindications*
   (a) Debilitated and advanced age
   (b) Heart failure which cannot be controlled (Advanced—Grade IV)
   (c) Presence of subacute bacterial endocarditis
   (d) Multi valvular disease which is disabling
   (e) Severe degree of mitral insufficiency
   Left ventricular enlargement etc

It is apparent that these indications and contraindications must vary with the individual case The report that patients with mitral stenosis live only an average of thirty-five months following the onset of hemoptysis and that the average duration of life following the first break or cardiac failure is four to six years[68] makes it apparent that in individual cases one must modify any set rules For example, auricular fibrillation is usually followed by the development of thrombus formation in the endocardium of the auricular walls Fifty per cent of these occur within the lumen of the auricular appendage[46] and many others are in the auricle It is an indication for the operation inasmuch as these thrombi can be surgically removed A recent careful study of mural thrombosis and embolism further confirmed previous reports that mural thrombi in mitral stenosis originates in the appendage in one-half of the patients [16,19,36,46] It appears that the reason for the thrombi occurring on the main wall of the atrium is the endocardial pockets or patches of MacCallum The site of one-half of these potential emboli can be eliminated merely by auricular appendectomy at the time of the commissurotomy

Ideally, one would select a patient in the younger age group with a high grade of mitral stenosis without advanced involvement of other valves The patients with severe mechanical obstruction with chronic fluid retention, large livers and evidence of poor myocardial musculature may be too advanced for the operation The symptoms of such patients are chronic weakness and fatigue, even on mild exertion, or orthopnea and cough with hemoptysis, palpitation, and pulmonary edema

**Consideration of Factors in Selection of Patients for Operation.**—Several factors must be considered The first and foremost is to determine how disabled the patient is at present Is there a good chance that the disease will progress and that the patient will move into a more advanced degree of the disease? Then the status of the patient's other organs and the relative ability to withstand the operation must be evaluated Several of the problems will be mentioned briefly

*Age*—There is sufficient experience accumulated for it to be said that age *per se* is no contraindication to the operation In this respect age should not interfere with therapy anymore than it should with the treatment of cancer of the stomach or uterus, provided there is a good chance that the operation will help the patient The older patient can be prepared to withstand the operation in most cases They do extremely well after operation, if in the first place the operation can help them

*Pregnancy*—This condition always makes the heart lesion worse and as the pregnancy advances the disease does likewise While interruption of the pregnancy is advocated in the early months by many, this is not a safe or innocuous measure either After the fourth month the patient probably can withstand commissurotomy better than she can an abortion

*Rheumatic Fever*—This condition depends on degree When there is a "hot," active infection the operation should not be performed That the operation can be done safely on many patients with some degree of active rheumatic fever is shown by the finding of Aschoff Bodies in half the amputated appendages after commissurotomy [27] Thus rheumatic fever alone is not a contraindication

*Subacute Bacterial Endocarditis.*—Where the operation can be postponed it should be delayed until six months after there is a bacteriological cure of the lesion. It is likely that the operation aids in preventing new attacks.

*Aortic Stenosis.*—This lesion is not a contraindication. It can be treated at the same operation time.

*Tricuspid Stenosis.*—This condition makes the operation more imperative for physiologic reasons. The two lesions may be operated at the same time or, if separately, the mitral stenosis should be corrected first.

*Tricuspid Insufficiency.*—This lesion improves after mitral stenosis is relieved.

## OPERATIONS FOR MITRAL STENOSIS

**Commissurotomy.**—The name for this operation was suggested by Durant.[illegible] This operation is an attempt to restore near normal mitral valve action. Its purpose is to divide the commissures in the line of their anatomic formation and at the site of their fusion which resulted from the rheumatic heart disease. Thus the direction of division will be anteromedially and posterolaterally. The division of the commissure must extend through the fibrotic tissue to normal valve tissue. In many only the lateral commissure has to be divided. In those in which arteriosclerosis is an added factor and there is marked calcification the medial commissure also must be divided. Division of the valve at any other point will cause increasing insufficiency. In addition if the scar is divided elsewhere a tear may extend the valve opening beyond the desired length. Scar tears roughly and, at times uncontrollably. It gives however best in the line of its formation and in the anatomic direction of the valve development. The procedure has for its function the relief of the stenosis without producing added insufficiency and therefore considerable experience is necessary before the surgeon can feel certain of his technic. The relief of stenosis especially in the calcified valves aids in the decrease of the insufficiency. This is so because in a calcified fish mouth-like valve the fixation of the leaflets and the immobility tend to produce insufficiency in itself. The release of these fixed structures thus permits the valve membranes to again function and close during ventricular systole. Over release or correction will negate this factor as will failure to open the commissures completely through the scar. Again it must be emphasized that surgeons who wish to do this type of work must practice repeatedly on autopsy specimens and as far as possible permit their inevitable technical errors to be eliminated on the postmortem specimen.

**Technics of Commissurotomy.**—The patient is positioned in the straight right lateral posture. The incision is made through the left lateral chest wall. Bailey prefers entering the chest through the fourth interspace. Other surgeons open the fifth or sixth interspace. Some elect to resect a rib. The author has seen no advantage and only a lengthening in the operative time by rib resection. The intercostal space is extended widely using a Finnochietto rib spreader. The pleura is opened. The heart is delivered and the pericardium may be injected with 2 per cent procaine both intra and sub-pericardially. The pericardium is opened ½ inch above or occasion

ally below the phrenic nerve, which is an outstanding landmark This incision presents the left auricular appendage which frequently herniates through this wound This appendage can be elevated by a Babcock type clamp (see Fig 19) Positioning the appendage with this clamp, a purse string suture is placed about the base of the appendage (No 0 or 00 silk on an atraumatic needle) The author's needle aids in this step (see Fig 23)



*Fig 18 —Anterolateral position for cardiac surgery Operative side is up Extension of arm removes it from the field and helps to swing scapula out of way*

Extraction of Thrombi —The external signs of a thrombus in the appendage or auricle often cannot be diagnosed The appendage is inspected for thrombus If it is suspected that there is a thrombus present, the appendage is cut off The appendage then is opened during an auricular systole and free bleeding into the operative field for one or more beats is permitted to flush out the clot At times when the appendage is cut, the clot can be seen and grasped and extracted by tension The base of the appendage should be held carefully on either side with Babcock type clamps before this maneuver is attempted The purse string suture is placed first in case of any untoward tearing or other accident which might occur at this time

Cerebral embolization occurs in 5 1 to 10 per cent of commissurotomy operations Since this was a large part of all complications in his series, Bailey advised an additional preventive measure.[6] At operation time the chest is opened in the fourth instead of the fifth interspace A silk ligature is placed around both the left carotid and the innominate arteries at their origin on the aorta These vessels are compressed during any and all

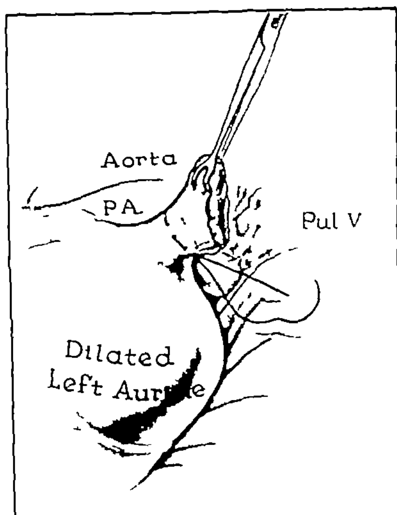



Fig 19 —Auricular appendage elevated with Babcock forceps Purse string suture of 00 black silk placed. Notice dilated left auricle Purse string suture should not encroach on auricle

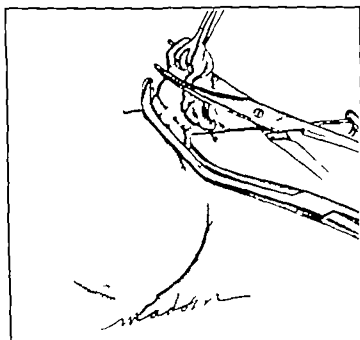


Fig 20 —Occluding clamp placed below purse string suture. Auricular appendage being amputated.

manipulations of the heart In 80 operations with this technic there has been no cerebral complication despite the fact that 26 had thrombosed appendices This change in technic is advocated to avoid this complication The base of the appendage is then clamped by a non-crushing type of hemostat (Sustinsky, Potts or Harken)

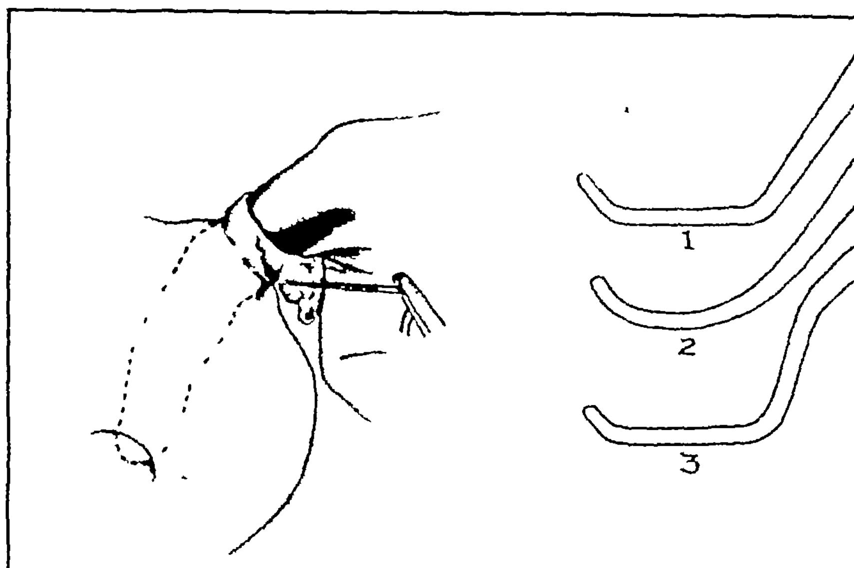



FIG 21 —End of appendage has been amputated Clamp released Index finger introduced into auricle and mitral valve as the purse string suture is tightened When the finger is withdrawn, the purse string suture is tightened and the clamp is replaced below the suture for safety during the oversewing Note various shaped clamps

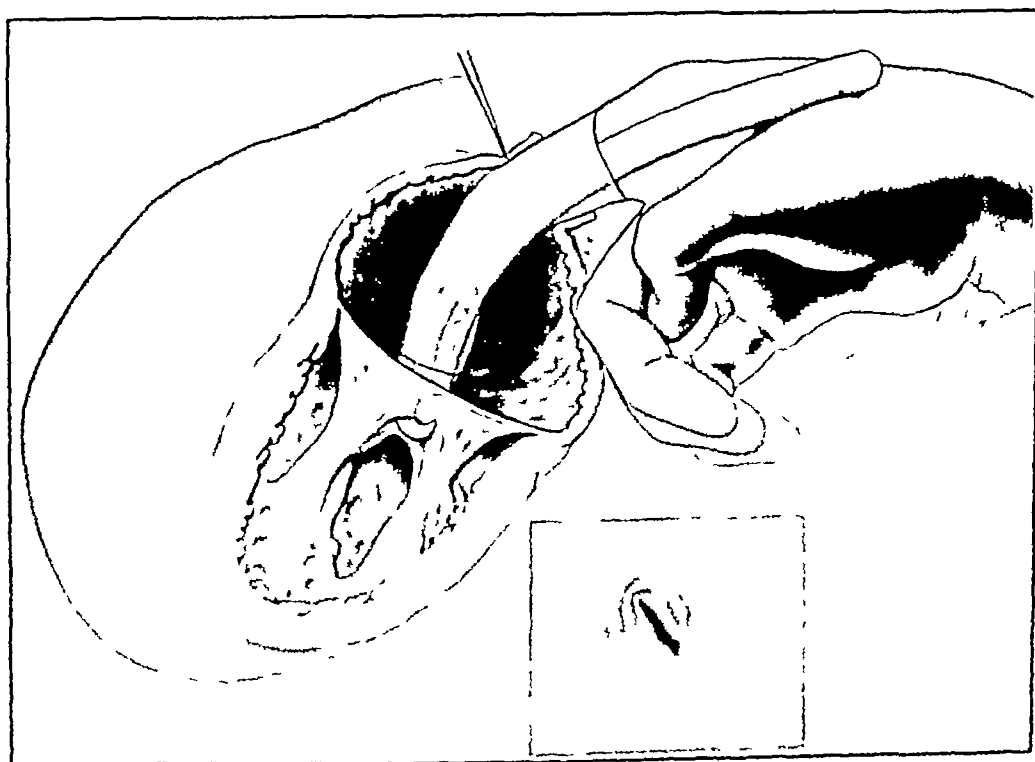


FIG 22 —Finger inserted into auricle and mitral valve with commissurotomy knife Knife can be used to increase the pressure of the finger In the calcified valve it may be necessary to divide the valve at the commissure Insert shows mitral valve

1 SIMPLE FINGER FRACTURE.—*Valvuloplasty*.—The gloved finger is introduced through the purse string into the auricle as the clamp is removed At the same time the purse string suture is tightened This controls bleeding The status of the valve as to stenosis insufficiency and arteriosclerosis then is determined If as usually is the case simple finger divulsion or fracture is sufficient to open the valve this maneuver only is performed The valvuloplasty is effected by sweeping the finger anteromedially and if necessary posterolaterally until the opening between the auricle and ventricle approximates one and a half to two fingers dilatation Too great a dilatation will be as detrimental or even more dangerous than ineffective breakdown of the scarred commissures The surgeon should have practiced on many post mortem specimens in order to have the feeling that results from lacerating the commissure in the anatomic line of union and at the site of its scarred pathologic obstruction With experience, the feel of the area will determine the effect and extent of the finger exeresis Following this manipulation its effect will be registered on the finger with contraction of the heart Any degree of insufficiency produced will be apparent by the expulsion of blood from the ventricle into the auricle and against the finger with ventricular systole Residual insufficiency can also be registered by the finger Ineffectual division of the commissures will be noticed by the experienced hand Whether the secondary changes following calcification have been or can be corrected also will be apparent

2 MECHANICAL FRACTURE.—In the *second type* of mitral stenosis the opening is fish mouth like but its edges are elastic Harken reports that 10 per cent of the stenotic valves he has seen fall in this category This elasticity precludes simple finger fracture as the valve gives with the finger pressure In this group if the fracture is possible it requires pressure as an aid In others excision of the commissure is a possible treatment

Following the development of the Brock valvulotome other instruments were added by Bailey Glover O Neill and Harken These consist of thin curved instruments which follow the natural curve of the index finger These instruments may be held against the finger by pressure or by a second glove This second glove is incised at the fingertip and hand web and the instrument is held in place between these incisions. The finger when introduced then into the mitral valve is reinforced with this flat instrument Such an instrument can be utilized to forcibly break the commissure particularly in the sclerotic cases In addition it is available to punch out a hardened area at the commissures resistant to digital or mechanical laceration.

3 EXCISION OR INCISION AT THE COMMISSURES.—In a certain proportion of patients (over 50 per cent according to Bailey[4]) with mitral stenosis there are anatomic reasons why finger fracture will not be successful In the calcific valve it may be physically impossible to break through the restriction with the finger or with a finger reinforced by an instrument In this group continued efforts to physically crack such a lesion may cause a serious tear beyond the normal valve structure The result may be an insufficiency that is more damaging than the original lesion In a second group the valve may be so elastic that it yields with pressure but never

divides at the commissures It has been shown repeatedly that the valve opening after operation must be at least 1 5 sq cm[28] Bailey believes strongly that the opening must be greater than this size The hard valve edge is cut with a flat guillotine type valvulotome which is modeled and fits the index finger The commissurotomy with the valvulotome is a complementary procedure to the finger inspection and fracture technic The incision must be made at the anatomic junction of the commissures This permits extension of the fracture to the normal muscle and through both the thickened basal ring and the secondary zone of fibrosis During the intracardiac maneuvers the anesthesiologist must observe the patient carefully for arrhythmias Therapy for such must be promptly instituted (see pages 39 and 59) After the intracardiac manipulations are completed the finger is removed from the appendage as the purse string suture is tightened and the clamp for the base of the appendage is applied The appendage is oversewn with 00 or 000 black silk after the purse string suture has been tied The needle devised by the author is of value at this stage and makes the oversewing easier The wound is then closed The pericardium is sutured lightly with 00 silk to prevent tamponade The pleura is not closed The chest is closed in layers as detailed under thoracotomy A catheter is left in the pleura This tube is aspirated after the airtight closure as the anesthesiologist inflates the lung The catheter is then attached to an underwater drainage The patient is kept well oxygenated during the closure All fluid accumulations in the chest are aspirated The patient is placed in an oxygen tent The vital signs are observed carefully Hemothorax, pneumothorax, mediastinal shift and cardiac arrhythmias and arrest are complications For safety, a trained member of the team should literally live with the patient for twenty-four hours The usual postoperative care of any thoracotomy wound is observed with greater diligence than usual Blood is replaced if needed but the heart is not overloaded The use of procaine postoperatively is individualized (see page 39)

**Immediate Aftercare of Patient After Commissurotomy.**—After operation the trachea and bronchi should be cleared and dried by aspiration Usually the reflexes have returned and the tracheal tube can be removed. If pulmonary edema is present, oxygen under positive pressure is given until it has been relieved This oxygenation will eliminate this edema in most instances The oxygen should be continued either by tent or catheter until such time as the respiration is stabilized and the patient is able to breathe without embarrassment

There is danger in overloading the circulation A limited fluid intake is important Most of the time it is best to remove the venous cannula Its presence often means its overuse If it is retained, the fluid should be at a very slow drip with careful measurement as to total volume.

These patients require sedatives but the drug should not be of the depressant type Most patients do well on minimal doses of morphine or barbiturates Pain interferes with movement and necessary cough and should be relieved They should be encouraged to move around freely in bed, to cough up any respiratory accumulation, and they should be permitted out of bed as soon as they have accomodated themselves to the intrathoracic manipulations

Bailey's[19] mortality of 9.4 per cent (56 patients in 592 operations on *all groups*) is a commendable figure. *Later figures of 5.8 per cent mortality indicate* the improvement with further experience.[45] The experienced surgeon soon acquires what Deaver called "the eyes on the tips of the fingers" so essential for this work.[1,24] The profession has accepted valvulotomy for the acquired cardiac lesions.[2,21,31,40,43,45]

**Author's Commissurotomy Needle.**—The insertion of this purse string suture successfully and satisfactorily is the key to the entire operation. It is a difficult technical feat since the heart is constantly moving with its heart beat and the operator has some degree of hand tremble if from no other cause than the gravity of the operation. The average needle must be grasped in the needle holder at right angles to the base of the needle,

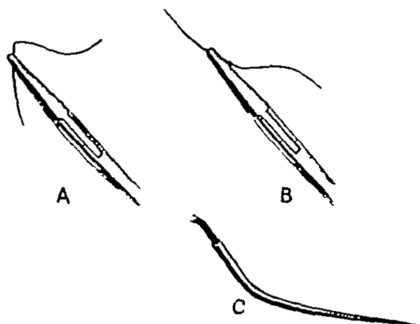



FIG 23.—Author's commissurotomy needle. Atraumatic ski-type needle. Base of the needle is made as a long rectangle (C). A and B illustrate how needle may be held by needle holder at any angle desired. (Pratt, courtesy J. A. M. A.)

*the part where the suture passes through the needle eye. In an effort to* hold the needle at an obtuse angle to permit the suture to be placed more readily, the surgeon often tries to catch the needle on a bias. The round needle tends to come out on the other side of the appendage before it has passed through both walls at the same level. The author, therefore, had a needle constructed for this difficult work. The needle instead of being conventionally round is of the "ski" type. It can be passed through both walls of the appendage at an even distance from the clamp. In addition, the site where the needle is to be grasped by the needle holder is made rectangular for considerable distance. In this way the needle can be grasped by the needle holder still at right angles to the needle base but at any angle to the object to be sewn from 0 to 180 degrees.[57]

**Variations in Technic.**—*Leak-proof Auricular Appendage.*—Thompson[64] created a valve in the auricular appendage by placing a purse string suture

at the tip and at the base of the auricular appendage This formed an outer and inner valve which permitted the introduction of instruments or finger without blood loss The inner set of sutures is placed parallel to the outer set and can be closed when the outer set is opened This work was done on dogs, but Thompson has used it on humans The auricular appendage in the dog is much longer than in the human and thus lends itself much better to such technic Where the auricular appendage in humans is anatomically similar, the procedure has possibilities One disadvantage is that in the placement of the second row of sutures close to the base the dangers increase if a tear occurs. The author has seen this accident on one occasion It is his policy to select the area on the appendage which is thickest and lends itself best to the insertion of the single purse string suture

**Prognosis in the Surgical Treatment of Mitral Stenosis.**—Patients first selected for this operation were terminal cases Forty-five operations in this group were followed by 14 deaths (31 1 per cent) While this mortality appeared prohibitive, at the same time, of 19 patients who refused operation, 17 were dead in a year (approximately 90 per cent) [41] Although this was not a true control group since many of these patients probably were dying, they do serve in some degree as a comparison The prognosis in this group is obvious Patients in group two and three should be considered for operation (see page 109). It is only a question of time until the cooperation of medical services will make this selection simple Unfortunately, at the present time many internists are skeptical They prefer to rely on digitalis and hope One must recognize that each attack of failure brings closer the inevitable day when the heart muscle can no longer recover In addition, the unhappy suffering of the patient drowning in his own fluids as they dam back from the left auricle into the lung should make each internist consider carefully if he is choosing correctly for his patient or merely following a precedent As surgeons, we are the first to admit that in the past we have overoperated Many patients are without their stomachs and more unhappy than they would have been on an ulcer diet regimen The sad pictures of the myxedematous patients created by the overzealous surgeon who removed a small thyroid enlargement are numerous Certainly many patients have right lower quadrant scars for innocent appendices On the other hand, part of the blame for this condition is the refusal often of many internists to call in a surgeon until the patient has been "medically cured" nearly to his death The majority of surgeons would like to have the decision as to surgery made by the conservative internist It is the failure or refusal of the internist to accept this responsibility which at times forces the surgeon's hand It is certain, for example, that all the blue babies were not born in Baltimore or Boston It was in these two cities, however, that the internists (pediatricians) and surgeons combined their studies to evolve a therapy which while surgically new and "radical" turned out to be conservative and lifesaving for the patient Thus with each new surgical technic must come the inevitable fight to determine if the method is "radical" or, in the long run, conservative

Only generalizations can be given in prognosis So many factors are involved that the result in each case must be individualized

Group I patients can undergo any operation

Group II can be operated in trained hands with a mortality of from 3 to 5 per cent

Group III patients will have a mortality of approximately 10 to 20 per cent in experienced hands This will be much higher in the surgeon who is operating on such patients only occasionally The surgeon's ability must be a factor in the selection of Group III patients until sufficient experience has been gained by many surgeons in many clinics. The significance of this discussion is to select the patient for operation before he enters a more advanced group That surgery itself will improve in the treatment of this lesion is certain Thus the occasional patient whose progress is arrested may be benefitted by delay In the majority however the added time just further depletes the cardiac musculature reserve on which operative success and subsequent well being depends This fact poses a challenge to the internist which he cannot refuse if his patient's true interests are his

**Other Operations for Mitral Stenosis.**—(a) VALVULOTOME METHOD THROUGH VENTRICLE.—Smithy[46] blindly tried to divide the stenosed valve by a valvulotome He first introduced it through the auricle and later through the ventricle because of hemorrhage from the thin walled auricle This method has been superseded by the others detailed This technic might have a place in therapy if no auricular appendage is present or had been amputated previously The ventricular approach also might be necessary if the auricular appendage tore during operation The blind valvulotome introduction would be replaced by the finger-guided instrument The value of the transventricular approach has been demonstrated by this method and also by the work of Brock in pulmonary stenosis[17]

(b) VENOUS SHUNT FOR ADVANCED MITRAL STENOSIS[52] (The pulmonary syndrome) —This procedure was developed by Sweet and Bland in an effort to produce an outlet for the high pressure in the pulmonary artery bed in advanced mitral stenosis As the stenosis increases the back pressure from the auricle into the pulmonary veins becomes great. It has been called the pulmonary syndrome As a result of this excess pulmonary pressure in an already overloaded system certain lesions and symptoms develop the most important of which is pulmonary edema Most of these patients die in a status of pulmonary edema literally drowning themselves The heart cessation is secondary to this edema Thus death is due to a pulmonary rather than a cardiac failure Post mortem examinations demonstrate this readily the cardiac musculature and coronary arteries relatively being undamaged

In an effort to overcome this pulmonary syndrome Sweet's operation has been successful in some instances

*Symptoms of the Pulmonary Syndrome*—The symptoms are those of cardiac failure and pulmonary edema. Pulmonary edema varies with the extent of the stenosis and is worse at night

*Hemoptysis*—Hemoptysis occurs in nearly every case

*Tachycardia*—Nature's effort to overcome this failure results in an increased rate of flow To these symptoms can be added the findings of hypertrophy of the right ventricle dilatation of the left auricle and the pulmonary artery and an increased blood volume on the right side of the

heart The bronchial veins dilate and it is from these vessels that the hemorrhage develops This may be minimal, "the so-called heart failure cells," or it may be massive up to 500 cc Large hemorrhages are followed by relief of the pulmonary edema, Nature thus performing its own phlebotomy

Bedford, Papp and Parkinson[14] have studied *Lutembacher's syndrome*[50] In this syndrome mitral stenosis is accompanied by an atrial septal defect These patients do not develop pulmonary edema, as the overflow can escape through the atrial defect and thus protect the lungs In this syndrome, more work is required of the right side of the heart because of this atrial opening, and the patients eventually die from cardiac insufficiency on the right side of the heart, but not from pulmonary edema Sweet noted that mitral stenosis occurs in 40 per cent of patients who have atrial septal defects He reasoned reversely that the atrial defect developed as an outlet for the mounting pressure in the patients with mitral stenosis Since 25 per cent of normal hearts have a minor degree of patency, this conception may be valid

Two possible methods of relieving this congestion existed. One was to make an opening from the left auricle or pulmonary vein into a systemic vein This included the hazards of an intracardiac or near cardiac approach to a high pressure area An extracardiac operation could be performed by uniting a branch of the right inferior pulmonary vein and the azygos vein The dorsal segment branch of the pulmonary vein was selected

*Technic (Sweet Operation—Pulmonary Vein-Azygos Vein Anastomosis)*[32] —The incision is made in the sixth intercostal space with or without rib resection The pleura is opened to expose the inferior pulmonary vein This inferior pulmonary vein is dissected free and the dorsal segment branch is liberated If this is short, the smaller branches near the hilus of the lung are divided to gain length The azygos vein is then dissected free by dividing the intercostal and other branches The superior segmental branch of the pulmonary vein is anastomosed to the azygos vein which has been divided 2 cc below the level of the pulmonary vein If sufficient length is not available, the esophageal, the hemi-azygos and the accessory hemi-azygos veins may be divided in addition to intercostal branches For the anastomosis Sweet used a vitallium tube of the Blakemore-Lord type which he believes reduces the possibility of thrombosis and the danger of laceration from suturing these thin-walled veins

*Comments*—This operation has been attempted too few times for final appraisal It may have a place in patients in whom the previously mentioned operations are not practical or where these have failed The difficulty of anastomosis of thin-walled veins under high pressure must be realized All who have performed portacaval or splenorenal shunts are aware of the technical problems which, at times, become insurmountable The technical ability of the originator is attested by Sweet's early reports on 6 successful operations The objections to the use of a foreign body tube for vessel anastomosis have been detailed elsewhere (pages 345 to 349)

In addition to thrombosis, the eventual rupture of the vessel at the point where the elastic wall meets the resistant tube makes many such anastomoses hazardous, although, without the cardiac propulsion present

on the arterial side this danger may be slightly less *The increased pressure on the pulmonary vein side may aid in preventing the thrombosis so frequently present when such tubes have been used in the past*

## SURGICAL TREATMENT OF MITRAL INSUFFICIENCY

Most cases of mitral insufficiency are associated with mitral stenosis *The heart and circulatory system can tolerate a regurgitation of perhaps* 1 ounce of blood per ventricular beat Such an insufficiency is compensated by a gradually enlarging heart In mitral disease during systole of the ventricle the mitral valve appears to remain open In such cases chordae tendineae probably hold the valve open due to shortening or attachment Mitral stenosis with fibrosis or calcification at the line of normal fusion of the valvular leaflets may fix and hold open the mitral valve during ventricular systole just as the mitral valve is inadequately opened during ventricular diastole Such patients have a paradoxical valvular dysfunction ' Ventricular contraction may pull the valve leaflets apart if the papillae have been distorted by disease

Mitral insufficiency if severe results in death During the primary systolic compression of the left ventricle blood is forced into the left auricle against pressure and secondarily against the blood returning from the lung by way of the pulmonary veins If not compensated pulmonary edema results from this back pressure

The presence of insufficiency is apparent during commissurotomy by the jet like ejection of blood against the finger in the auricle during ventricular systole The heart can tolerate about 20 cc of regurgitated blood per ventricular pulsation and this insufficiency only for a time

(*a*) **Vein Valve Grafts** —The first work with surgical repair of mitral regurgitation was reported by Murray in 1038 [32] When he incised the lateral mitral valve producing regurgitation all of the dogs so treated *died He then inserted an inverted vein graft completely through the left* ventricle by means of a cannula This produced a flap valve which partly acted as a tamponade for the mitral insufficiency during ventricular contraction Two of his 8 dogs survived Bailey [4] *et al* repeated these experiments These operations were then tried on humans While the graft functions for a time it gradually becomes a fibrotic band and loses its resiliency and thus its value function Inadequacy of such measures is further proven by the work of Templeton and Gibbon [45] Their efforts to suture pieces of pericardial tissue as fresh valve grafts were followed by fibrosis and dysfunction Thus any tissue that loses its normal blood supply has been proven inadequate for valve graft.

(*b*) **Pedicled or Tubed Grafts for Mitral Insufficiency** —Strips of pericardium with their normal nerve and blood supply retained have been utilized for mitral insufficiency The same requirements for operation as in mitral stenosis exist The patient must be in a state of cardiac compensation

*Technic* —With the patient in a right lateral position the fifth left interspace is opened from the vertebrae to the mammary area The pleura is incised and the pericardium is then opened posteriorly to the left phrenic

nerve A purse string suture is placed around the left auricular appendage and a clamp placed at the base of the appendage. After the auricular appendage has been incised, the index finger is inserted as the purse string is tightened The amount of regurgitation, the presence or absence of stenosis, the character and sclerotic nature of the valve is then determined If stenosis is present, it is corrected, as in mitral stenosis If an insufficiency exists, a pericardial flap is prepared, preserving the blood and nerve supply Excessive fat in the flap is excised The pericardial flap is folded so that the epicardium is external The index finger of the right hand is passed through the orifice and then into the ventricle The wall of the ventricle over the anteromedial commissure of the mitral valve is located The eye end of a probe through which is treaded a suture which is attached to the pericardial flap is passed through a small incision in the ventricular wall and grasped by the intracardiac finger It is guided in close proximity to the ventricular aspect of the mitral orifice, to the posterior commissure and its chordae tendineae The probe is then pushed to emerge lateral to the posterior aspect of the left ventricle avoiding the coronary vessels When the probe emerges, its suture is grasped and the probe is then drawn back out of the heart The posterior suture thus pulls the free end of the graft into the left ventricle and then out of it posteriorly This graft then is sutured to the outside of the ventricle The free end of the graft is permitted to retract into the ventricle and where its tip remains it is sutured to the epicardium of the ventricle This retraction provides the slack for valvular action The degree of movement of the graft and its valvular action can be ascertained by the intracardiac finger If the graft is of the right size and properly placed with sufficient redundancy, it will tend to tamponade the regurgitation If the graft does not function well, it is probably because it has become fibrotic or the technic of the surgical procedure was not satisfactory Bailey[4] *et al* considered that the success of the procedure depends upon whether the graft is over the valve, is not tight, and that it is inserted directly through the chordae tendineae which are at either end of the mitral opening The surgical correction of this lesion has not been successful so far

## AORTIC STENOSIS

The early work on surgery of this type has been discussed (see pages 102 to 104) Tuffier's successful relief of an aortic stenosis by dilatation of the aortic valve occurred in 1914 and the patient lived at least until 1924 [62,64]

Aortic stenotic lesions are difficult to treat surgically since the danger of aortic insufficiency is greater than that of aortic stenosis When the patient develops an aortic stenosis slowly, the heart compensates for it by a hypertrophy and thickening of the left ventricle, but the ventricular chamber does not enlarge The only time the chamber enlarges is when there is an associated aortic insufficiency When aortic insufficiency is suddenly created by the surgeon, the thickened ventricle is less able to dilate to accomodate the extra blood present in even a slight aortic insufficiency

In the autopsy room a stenotic valve can be dilated to break apart the commissures which have been fused by the rheumatic disease Any dilating device will accomplish this method similarly to that occurring in the mitral valve The problem is placing the dilator atraumatically at the correct site and not injuring the valve in dilating it

**Pathologic Physiology** —Aortic stenosis in general, is caused by rheumatic heart disease This results in a thickening and fusion of the valve cusps which then become hardened and rolled out Nearly all of the valve leaflets become rigid and may contain calcium plaques The available literature supports the rheumatic origin of most of these cases and the calcium deposition is probably only the end result Some feel that arteriosclerotic aortic stenosis is fairly common The commissures which are the fused valve edges are indurated and this hardness may extend into the aortic wall The fusion may not be equal in the three commissures and this causes dislocation of the orifice The two anterolateral cusps fuse most often and this transforms the valve into a tricuspid one The coronary arteries which open 1 cc above the two anterior cusps rarely are involved

As a result of the stenosis the left ventricle can expel only a small amount of blood. The systolic pressure although not high is sustained longer than normal The diastolic pressure which is high decreases the pulse pressure The ventricle s contraction is increased as its load strikes the closed aortic valve The coronary arteries do not obtain as much blood flow at a time when they require an increased amount This results in the angina The symptoms are those of diminished output Mild exertion produces dizziness and faintness. The supply and demand ratio is distorted as is the coronary flow Sudden death is frequent after even mild exertion due to the disproportion between the amount of blood available and that required Unlike other valvular diseases the response to medical therapy is negligible This is due to the fact that the coronary arteries must be filled as their flow is needed for the hypertrophied left ventricle

1 *Valvulotome or Punch Resection* —Smithy s[40] approach to the problem of aortic stenosis was a punch excision of a section of the malformed valves He believed that the regurgitation thus produced could be tolerated by human patients. His punch instrument was inserted through the ventricular wall hemorrhage being controlled by a purse string suture Other attempts to utilize the aorta as a passageway for the punch were unsuccessful This method of therapy has been discarded The diseased heart is unable to compensate for the resultant regurgitation While over 80 per cent of the dogs die when a cusp is divided the human heart with aortic stenosis cannot survive such excisions at all

2 *Artificial Aortic Valves* —Hufnagel[44] Campbell[31] and others have made attempts at construction of aortic valves This work is early incomplete and unproven but is praiseworthy and should be continued The problem experimentally is difficult The experimental animal does not have human aortic stenosis nor can it be produced The human heart specimens available are the advanced ones and therefore beyond the stage when surgery might have been attempted This work at the present time must be considered experimental The possibility of interference of the nearby coronary arteries must be always considered in the placement of

any artificial valve If such a valve is constructed, it is our belief that it must be of a non-rigid material which will give with the propulsion of the pump's contraction

3 *Valve Grafts* —The attempts of Bailey[7] *et al*, Murray[51] and others to make valve grafts are experimental but hopeful The early efforts to use analogous and homologous grafts were not satisfactory Bailey's efforts to pass an inverted vein graft had a prohibitive operative mortality from hemorrhage The experiences with valve grafts as reported by Gross *et al*[39] do not indicate that the future of such efforts will be fruitful Experience, however, makes one hesitate to discard any measure that has had even a single success

4 *Aortic Valve Homografts* —Lam *et al*[49] have experimentally used valve implants for the aortic valve While half of the animals died, in half of the remainder the valve appeared to function

5 *Other Analogous Grafts* —Free grafts of the aorta, with or without polythene reinforcement, have not been satisfactory Aortic regurgitation apparently is an inevitable complication These experiences apparently refuted the contention of Smithy and Parker that the human system could survive a small amount of regurgitation if the stenosis was relieved

6 *Direct Division* —Brock's method for the direct division of the pulmonic valve in congenital pulmonary stenosis is now an accepted and advantageous treatment of this lesion (see page 80) In pulmonic valve stenosis the tricuspid valve becomes fused before birth, forming a cone-like tiny orifice Through this opening only a small amount of blood can pass to the lung The blood usually passes through another congenital defect into the systemic circulation Brock's operation consists of making the stenotic tricuspid valve into a bicuspid one by dividing these fused cusps into two halves He uses a diamond-shaped knife inserted through the right ventricular wall

Bailey[7] *et al*, perceiving that the aortic valve was similar anatomically to the pulmonic valve, attempted the same procedure Despite anatomic correctness of the operation, a regurgitation resulted with which life was incompatible This procedure may be developed later, but cannot be recommended at this time in view of the reported dire results The cartilaginous or bone-like structure of the aortic valve may tear at the line of least resistance, the anterior lip, and thus a regurgitation incompatible with life will be produced

7 *Retrograde Dilatation* —Dilating the valve as first extra-aortically performed by Tuffier remains as a possibility It is doubtful that the aorta can be invaginated safely, especially if it is calcified A direct attack on this valve by the introduction of an instrument through the carotid or other branches off the aorta is a future possibility In such an operation, an instrument is introduced through the lumen of the right common carotid artery The instrument follows the innominate artery and the ascending aorta until it reaches the aortic valve Such a position can be recognized by the transmitted cardiac impulse and an increase in the systolic thrill The dilatation part of the instrument is then utilized by the opening of a dilating screw in the handle

In some animals this has been successful The possibility of creating a false passage due to the difficulty of entering a stenotic valve from above exists and a fatal case is reported by Bailey[4] *et al* Entering the valvular regions through an instrument like a transurethral cystoscope has been considered Such an instrument could reach the site of obstruction if it were inserted through the carotid artery Direct surgical vision with resection and coagulation if necessary appear feasible technically It might remove the hazard of rupture due to an eccentrically placed orifice

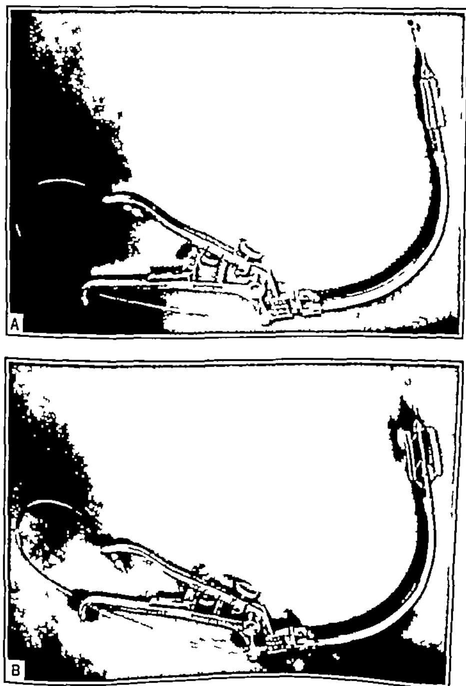



FIG 24 —Bailey's dilator for aortic stenosis. *A* Wire probe is introduced through the valve and the dilator follows the probe as a guide *B* Shows dilator opened (*J* ~ Donaldson, Chatham, New Jersey )

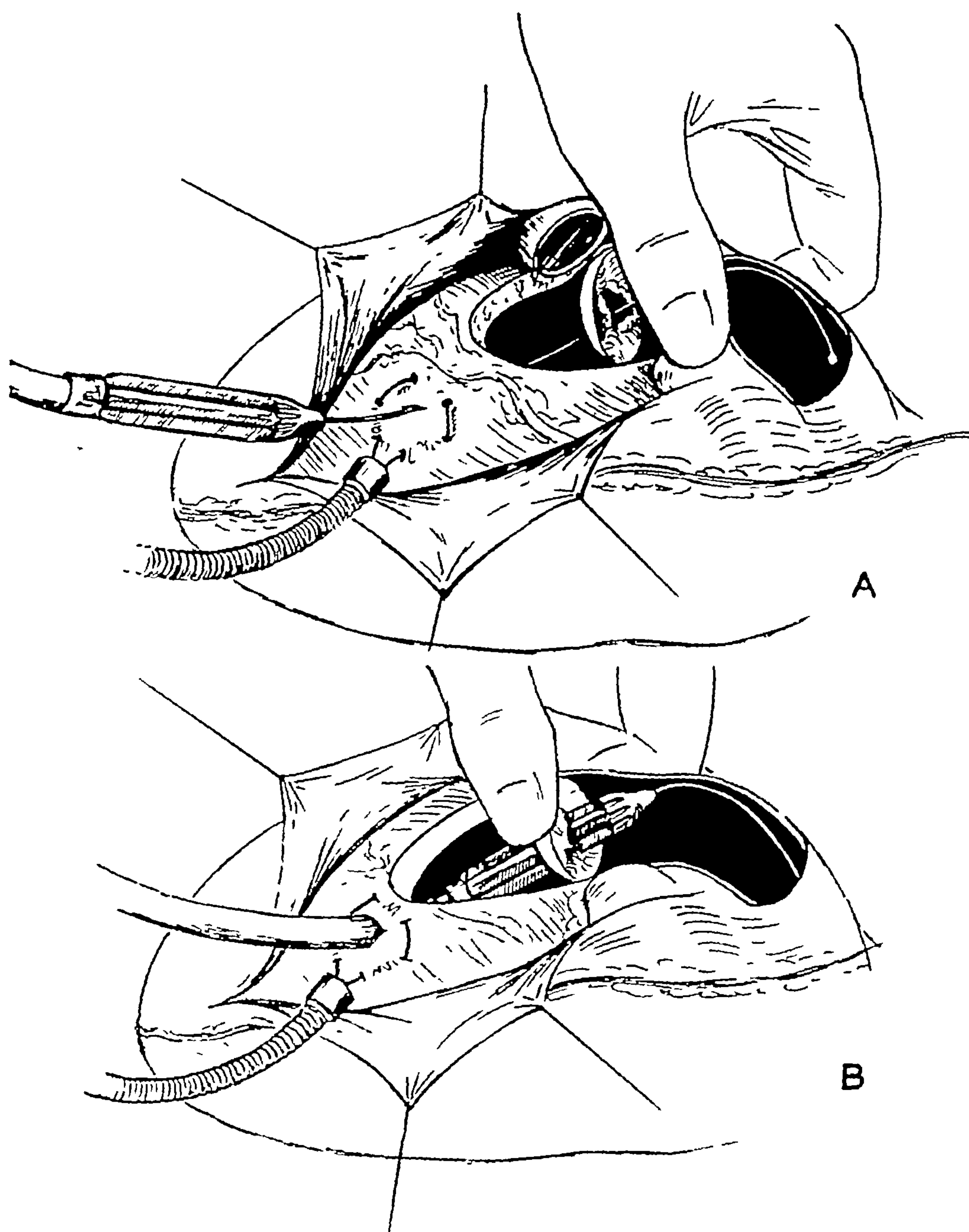



Fig 25 —Operation for aortic stenosis Dilator follows probe through valve like an esophagoscope follows guide string (Courtesy of C P Bailey, Philadelphia )

8 *Dilatation of Aortic Valve Through Ventricle* —After trying various types of blind dilatation with "umbrella-like" dilators, Bailey and his co-workers[7] have been able to overcome aortic stenosis to some degree The technic is as follows

A 5 cm transverse incision is made in the pericardium just above the apex of the ventricle A heavy purse string suture of silk is placed deep in the left ventricle, avoiding the phrenic nerve and the coronary artery A second pericardial incision is made at the base of the heart to expose the aorta A small incision is made in the center of the purse string A wire with a round guide is passed through the ventricle and the aortic valve into

the aorta where it can be felt with the other hand on the aorta The dilator is then fed over the wire like one passes an esophageal dilator over a string which has been passed through the obstruction The parallel dilating bars are opened when the instrument is directly at the stenotic lesion The parallel bars thus separate the commissures The instrument is then reduced and withdrawn Hemorrhage which has been controlled by the purse string sutures is then stopped by interrupted sutures The purse string suture is then withdrawn

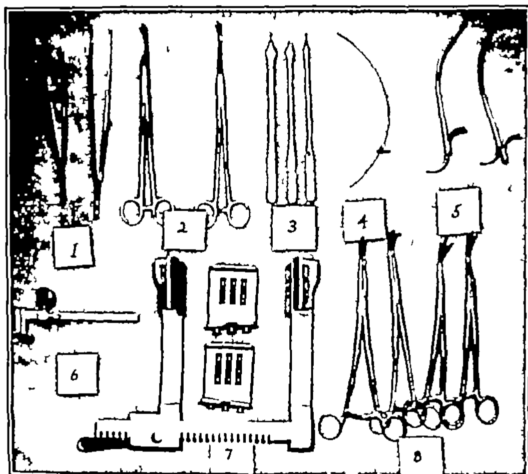



FIG. 26 —Special instruments for cardiac surgery (1) Long Mayo-type forceps (2 and 8) various sized and shaped clamps for occluding the base of the appendage (3) universal valvulotomes (note dull probe end) (4) Harken type valvulotome (5) valvulotomes after Bailey Glover and O'Neill (note that 4 and 5 valvulotomes fit hand and finger) (6) rib approximator (Bailey) (7) Finnochietto rib spreader (Note various sized retractors )

## COMBINED MITRAL AND AORTIC COMMISSUROTOMY

If aortic and mitral stenotic lesions coexist it is best to relieve both obstructions at the same time. Deaths have followed commissurotomy on the mitral valve when aortic stenosis is present due to an imbalance created after the mitral valve is repaired with more blood entering the left ventricle When relief of both lesions is to be performed the mitral one is attacked first. The left common carotid artery and the innominate artery are temporarily occluded to prevent emboli to the brain (see pages 112 to 114)

The opening of the aortic valve need not be too great to improve the condition The heart compensates for aortic stenosis in some degree, probably until the opening is 10 per cent the size of normal A 50 per cent enlargement is all that is necessary Bailey's mortality rate of 36 per cent by old instrumentation dropped to zero in 9 patients with the new dilator method described Two-thirds of these patients had both aortic and mitral stenosis lesions relieved

## TRICUSPID STENOSIS

This lesion is rare but similar in origin to mitral stenosis The two occur frequently together Both lesions can be surgically corrected at the same time

## AORTIC INSUFFICIENCY

This lesion has been surgically corrected at least partially The efforts of Campbell[21] and especially Hufnagel[44a] to devise valves are praiseworthy If successful, autogenous valves probably will be the answer

## TREATMENT OF HEART FAILURE BY VENA CAVA LIGATION

In congestive heart failure, the reduction in the amount of circulating media is followed by immediate improvement Phlebotomy in the acute mechanical failure has saved innumerable lives. In a like manner, the reduction of blood pressure in the right auricle and ventricle will improve, at least temporarily, the symptoms of congestive failure In the patients who do not respond to any conventional type of therapy for their congestive heart failure, a reduction in the pressure in the right side of the heart has been achieved by an inferior vena cava ligation This was suggested originally in 1948 by Cossio and Perianes[23] Other surgeons, particularly in South America, have produced additional data Of the 81 patients out of 90 who survived the operation, 63 showed improvement. It is noteworthy that medical therapy was continued in each instance The most favorable response occurred in patients with signs of thrombosis of the legs The possibility that sources of emboli were thus removed has to be considered

The operation when performed has an operative mortality rate consistent with surgery in any patient with congestive heart failure The anesthesia should be regional or spinal and the ligation should be performed in continuity The application of this technic is limited. The technic of inferior vena cava ligation is detailed on pages 637 to 639

## THE BALL VAL[illegible]ART

Rarely an intracardiac thrombus i[illegible]fficient size to act as an obstruction to the passage of blood [illegible] valve The thrombus

may be sufficiently round to only partly or irregularly obstruct the blood flow. Such a condition is inconsistent with life but it may permit the circulation to continue for some time. The *symptoms* depend upon the degree of obstruction. They are hypotension, cyanosis, dyspnea, decreased pulse volume and evidence of a failing general circulation. Emboli may occur. The *treatment* problem rarely is raised because the diagnosis is not made in most cases. The surgical possibilities of successful relief exist. Through the auricular appendage the auricle can be reached. The clot may be broken up and extracted or removed in one piece. The possibility of embolism during such a manipulation must be considered. Temporary constriction of the vessels to the brain may prevent a fatal embolism, as in a thrombosis accompanying mitral stenosis. The auricular appendage should be oversewn. Such patients require anticoagulant therapy for an indefinite period.

## SUMMARY

Thus it is apparent that the operative treatment of acquired valvular lesions is no longer speculative, experimental or impossible. The large hiatus in cardiac surgical advance produced by failures in certain technics appears to be over. Animal surgery is essential to the development of these technics. The carry-over of such surgery to clinical application to human beings can never be ideal. Patients may die in the attempt to cure them surgically and in the development of the technic. We must realize that most of these patients are doomed regardless of therapy. We must also recognize that advances are halted by such conservative attitudes as restrict surgical intervention to the patients who are hopeless. Many patients have been referred for surgical intervention because all else has failed. Such individuals often will die from the suggestion of an operation, *the preparation for the transfer to surgical service or the necessary anesthetization.* Deaths of such individuals should not be credited to the surgical operation. When the internist and surgeon with the collaboration of a cardiac physiologist will select the patients jointly for operation the problem will be settled. Surgically it is necessary to have patients with cardiac reserve. Those selected should be patients in whom it is obvious that repeated attacks of cardiac failure similar to those already encountered will be fatal. Procrastination until the cardiac reserve is inadequate will lead to a large and prohibitive mortality.

It will become the duty of the internist to select the patients who require this therapy at the time when they can tolerate it. This requirement must be accepted by the medical specialist. It is the hope of the cardiac surgeon that the internist will become as prompt and adroit in his reference of such patients at a time when they are amenable to surgical intervention as did his older brothers in the selection of patients for operation when an equally controversial medical and surgical problem was presented by the new-fangled diagnosis of appendicitis.

Over 500 patients have been operated in Bailey's clinic.[4]

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Chapter

# 11

# SURGERY IN THE TREATMENT OF CORONARY OCCLUSION, AND THE CARDIAC PATIENT AS A SURGICAL RISK

## SURGERY IN THE TREATMENT OF CORONARY OCCLUSION

ONE of the great challenges to the medical profession in general and in particular to the cardiovascular surgeon is the replacement of or the ancillary addition to the coronary circulation of the heart. Coronary artery disease with its resultant myocardial infarction remains the greatest "robber" of our time. This thief, while no respecter of any class, appears to select in a predilective fashion the most productive and promising of our younger adults. He strikes suddenly and often without warning. The examples of his work can be best exemplified by members of our own profession. It appears certain that in the future the care of these patients can be made a positive one rather than relegating them to the medical supportive measures now available. Last year 200,000 Americans died of coronary artery disease.[41] The relative frequency with which young people die of coronary disease has been emphasized.[13,14] The ratio of men to women with coronary disease under forty years of age is 24 to 1. Fifty per cent of the men, or 25 per cent of the entire population, are possible candidates for coronary heart disease.[44] Efforts to preselect those who will develop the disease and fit them into a profile pattern have been made. Sufficient study has not permitted incontrovertible deductions to be drawn. It is known, however, that patients with a hereditary coronary background with a high serum total cholesterol level, with an increased serum uric acid figures and an elevated cholesterol/lipid phosphorus ratio are more likely to develop coronary heart disease at a younger age. It is a common cause of sudden death in young soldiers.[13] The incidence increases with age and occurs most often in cold weather. The variability of the disease is characteristic. In one, anginal pain may give warnings for years, in others a sudden and apparently primary attack is fatal. Our inability to differentiate the type probably interferes with therapy. The extent of the thrombus and the collateral blood supply determines the outcome.

Angina was known even before its classical description by Heberden in 1786. His description entitled "Some Account of a Disorder of the Breast," indicated how lost the early physicians were in the early diagnosis.[24] Jenner, having seen dissections of the heart, recognized that the coronary arteries were basic in ischemic heart disease. He refused to

write about his beliefs for fear of alarming his good friend John Hunter, who had the lesion[24] While Laennec thought angina was a neuralgia or rheumatism, he is to some extent responsible for the therapy which has to do with blocking the nerves to the heart

The treatment of coronary heart disease primarily is medical and will remain so until the surgeon can show an improved mortality, at least in selected instances

**Prognosis in Acute Myocardial Infarction.**—Statistically, there has been a correlation between age and survival in the attacks of myocardial infarction The prognosis has been very poor in those over fifty-five[19 39 43 49] Approximately 25 to 30 per cent of the patients die with the attack[43] Of those who recover, 40 per cent make a clinically complete recovery Another 25 per cent recover, but have some angina Twenty-five per cent live ten years, and 40 per cent live five years[32] The higher mortality in the acute attacks in those over fifty-five is due to a more serious attack which coincides with myocardial deterioration The prognosis in the individual attack probably is the same in the elderly patient as in the younger one[4] Conversely, the very young individual frequently has a bad prognosis because he has developed no collateral circulation

A disease in which the mortality is great is always a challenge to the surgeon. Thus, the hopelessness of congenital heart disease under a medical regimen stimulated surgical efforts Innumerable examples, such as the successful treatment of aneurysms, pulmonary tuberculosis, and others, have developed even in our own time In a similar fashion, coronary artery disease with its high mortality and disability rates and the severity of the pain symptom itself causes the surgeon to hope that further research will develop some modality for the relief of this distressing condition

No effort is made in this book to detail symptoms or diagnostic measures in coronary heart disease These are available in general medical texts

The rapid advance in the surgical handling of all lesions in the thorax makes possible surgical intervention in certain selected cases This intervention has been promoted by the modern anesthetic improvements, the antibiotic drugs and better vascular suture technics

In the past surgery has been used in coronary occlusion with the following aims

(1) To relieve the pain of angina pectoris by means of (a) sympathetic nerve blocks and (b) sympathectomy

(2) To establish artificial collateral circulation to the heart by means of (a) cardio-omentopexy, (b) pectoral muscle transplant to the heart, (c) cardiopneumonopexy, (d) production of pericardial adhesions, (e) vein transplant from aorta to coronary sinus, (f) efforts to connect a systemic artery to the cardiac muscle or a coronary artery

The efforts today are directed toward the replacement of the coronary arteries themselves by other arteries not needed by the patient While still in the experimental stage, these potential aids show sufficient promise to be considered in the therapy of patients whose future otherwise is hopeless Other therapy includes direct thrombectomy of the coronary artery and tube grafts with skin

**Relief of Pain.**—(*a*) INJECTION OF SYMPATHETIC GANGLIA —The pain of angina pectoris results from a disproportion between the oxygen demand and the oxygen supply of the myocardium It can result from (*a*) an arterial spasm which is usually secondary to the disease (*b*) a coronary arteriosclerosis or inflammation (*c*) severe anemia when insufficient oxygen is carried to the myocardium or in gas poisoning (carbon monoxide) (*d*) mitral and aortic valve disease when the minute volume output of the heart is too low (*e*) when the mean aortic pressure is too low as in aortic insufficiency or in tachycardias (*f*) when there is a disproportion between blood supply and the hypertrophied heart muscle The terms cardiac pain heart pain coronary insufficiency, and angina pectoris can be used interchangeably The pain of angina pectoris is extremely severe It may be likened to that of a foreign body in the eye transferred and expanded in severity to the entire thorax Pain in itself, by a sympathetic syndrome may be the stimulus in continuing the anginal process, thus establishing a vicious circle

The cardiac nerves in the thorax provide direct communication between the heart and the upper five or six thoracic ganglia In most instances the sympathetic chain at D-2-3-4-5 has a specific effect on the cardiac rate Chapman *et al*[11] demonstrated that interruption of D 2-3-4-5 on both the right and left sides lowers the basal pulse rate and decreases the cardiac acceleration which accompanies exercise Interruption of this pathway may provide relief of the symptom of pain in as high as 60 per cent of the patients This possibility should be considered in those in whom pain is the only symptom and where pain is the factor in continuing the process

To surgically interrupt these connections in this poor risk group remains a formidable procedure and because of the cardiac status in many cases is not feasible The chemical interruption of these sympathetics with novocain and alcohol or other drugs is possible and most of the time it is successful. There is a definite risk, but where the danger from the angina is great this must be accepted The effect may be temporary but it can be repeated as often as is necessary One must remember that the pain of angina pectoris is one of Nature s warnings and a guard against the overuse of the heart. The relief of this pain destroys this warning and thus carries some risk. In some patients this relief cannot be offered because of the lack of self-control of the individual and his activity when there is no pain When the pain is relieved all sense of warning may be eliminated

The technic for interruption of the sympathetics for the relief of anginal pain was first used and standardized by Leriche[24] and Jonnesco[25] The procedure probably reduces the possibility of sudden death from the motor spastic response set up by the pain itself which follows the attack.

*Injection Technic* —The injection is made from the second dorsal to the fourth or fifth dorsal ganglia. This means that the nerves are interrupted from the first to the fifth ribs The needles are inserted paravertebrally and advanced carefully until they are in contact with the vertebral wall near its anterior surface

Since the pleura is adherent to the vertebrae a pneumothorax is a possibility To reduce the danger of tension pneumothorax a drop of

novocain can be placed in the needles as the stylets are withdrawn If the needle tips are in the pleura, this drop will be aspirated by the negative pressure In such event, the stylets can be replaced and the needles removed to a safe area

Two cc of a 2 per cent novocain solution are injected slowly and the patient's clinical response is observed. If the position of the needle is correct, the patient's symptoms will be relieved Three cc. more of novacain then may be injected If there are no untoward reactions, 4 cc. of absolute alcohol (or 95 per cent alcohol)[50] are then injected

Complications of the procedure may occur Due to the irritating effect of alcohol an *intercostal neuritis* may be troublesome and persistent Pleurisy with or without effusion has occurred The possibility of intravenous or intradural injections should be eliminated by the technic A pneumothorax, always a danger and technically possible, should not occur if one is careful and trained in the technic described A vago-vagal reaction with cardiac arrest is a rare complication It may require cardiac massage or cardiac muscle shock to break the reflex This demands immediate diagnosis and therapy or death will follow. The results of this injection therapy are good in 75 per cent of the patients [50]

(*b*) Sympathectomy —At times, a permanent surgical interruption of the sympathetic system may be considered if the temporary response is good For a sympathectomy to be successful, the second to the fifth dorsal ganglia must be removed Since some sympathetic fibers may arise higher in the cervical plexus or below the fifth dorsal ganglion, the operation is not always successful Cervical sympathectomy alone is not sufficient Flothow,[14] Lindgren and Olivecrona[31] advise removal of the stellate and the first four dorsal ganglia Rainey,[39] Utterback and Klemme[45] remove only D-2-3-4 Their reported results indicate that this latter operation is as satisfactory as the one which includes the cervical and stellate ganglia The sympathetic nerve supply of each ganglia in this area is unknown or inexact Overlapping may be the cause for some of the unpredictable results of denervation The question of whether vasoconstricting fibers at times may be included with motor fibers is a mute one [26,50] Should this be so and a sympathectomy performed, obstinate and constricting effect on the coronary vessels might follow Fortunately, this is a rare occurrence

The possibility of sensitizing the part to circulatory sympathin or adrenalin, as happens in the upper extremities when a postganglionic operation is performed, must be considered. Our understanding of the sympathetic innervation of the thorax and the upper extremities, even after all the work that has been performed, is far from complete and is retarded by the comparison with our understanding of the action of the sympathetics in the abdomen and in the lower extremities Inability of the patient to withstand the position necessary to perform the sympathectomy and/or the anesthetic may play a deciding role in whether or not to employ sympathectomy

**Establishment of Collateral Circulation to the Heart.**—Five surgical procedures have been advocated in attempts to introduce a new blood supply to the heart to replace that which is lost as the coronary vessels

become occluded These methods take advantage of the efforts of the heart to develop a collateral circulation They are formidable procedures, attended by high mortality rates

Before employing any of these technics one should bear in mind that coronary occlusion is not necessarily fatal if it is not acute, a point brought out by Leary and Wearn[24] The heart itself develops some collateral circulation when stress is put upon it by occlusion of coronary vessels. Hudson Moritz and Wearn[23] demonstrated collateral communications between the heart and the vascular fat at its base. The good results of employing antithrombotics in the acute phase and on a long term ambulatory basis have been proven efficacious[14] This may alter surgical attempts to provide blood

(a) CARDIO-OMENTOPEXY —O'Shaughnessy[33] was the first to try cardio-omentopexy The omentum is an unusual therapeutic agent Thus we see it wrapped firmly around a fibrotic uterus trying to supply blood surrounding a twisted pedicle of an ovarian cyst aiding in the localization of infection after a bowel resection or enveloping an appendix or a perforation of peptic ulcer When one tries to separate the omentum from these organs it is densely adherent It is evident that the blood supplies of the two parts have intermingled The mortality rate of this operation was too high compared with the beneficial results and it can be advocated no longer

(b) PECTORAL MUSCLE TRANSPLANT TO THE HEART —This operative procedure was advocated by Beck[2,4] in 1936 It was a direct attempt to treat the disease rather than the symptoms The chest wall is opened, the pericardium incised and a pedicled flap of the pectoralis major muscle and its vascular fat is sutured directly on to the heart wall In his first group of 12 patients on whom he operated Beck[3] recorded a mortality of 50 per cent This dropped to 15 per cent in his later series of 13 patients

The high mortality and the equivocal results have led nearly all surgeons to discard this procedure The incorporation of a direct artery or vein transplant with the muscle offers better possibilities Other substances besides the muscle may carry blood to the myocardium

(c) CARDIOPNEUMONOPEXY (LEZIUS) —The principal of this procedure was to permit the heart to become adherent to the lung Lezius had reported that if the heart became adherent to the lung sufficient collateral circulation at times developed so that when the coronary artery was ligated the animal did not always die

Carter[10] and his associates used asbestos powder to produce vascular adhesions between the heart and lung They were able to cut the mortality in dogs following a ligation of the anterior descending branch of the left coronary artery to nearly 1 in 3 if the cardiopneumonopexy had been performed Reimann and his associates[41] established the communication by suture graft Others have used similar measures The autopsy reports of Nature's frequent effort to utilize this measure of vascularization of the myocardium from the lung source indicate its feasibility The ease with which the body accommodates itself without an entire lung makes this source of blood supply a potential one for future investigators in this field *This operation as originally conceived is not being used at this time*

(*d*) PERICARDIAL ADHESIONS —No longer do we consider the adhesions of pericarditis as a complication of coronary disease in those patients who survive It is now felt that these adhesions are an attempt of the heart at healing rather than a complication Thorel[57] reported a patient with chronic adhesive pericarditis in which both coronary arteries were long occluded without any symptoms of coronary disease during life

That such adhesions may be produced to increase the blood supply is the basis of attempts to establish collateral circulation Not only may this yield a new way for supplying blood to the heart, but perhaps it may be a bridge for collateral circulation to develop between the right and left coronaries when one of them is blocked, as shown by Beck, Tichy, and Moritz[6] Such collateral circulation should improve with adhesions between the heart and its covers While half of the dogs die when the anterior descending branch of the left coronary artery is divided, this figure can be halved after cardiopneumonopexy

Thompson used talcum powder as early as 1939[44] He still uses powdered magnesium silicate Powdered beef bone was placed in the pericardial cavity by Beck[5] More recently we have studied the effect of talcum powder and its irritating action It is known that some of the dense adhesions encountered after abdominal surgery are linked directly with accidental tearing of rubber gloves with a release of the enclosed talcum powder irritant Other substances which have an intense irritative action are certain types of polythene or certain chemicals (dicetyl phosphate) used in the preparation of polythenes Polythene rapidly will set up an irritative exudative reaction Most such reactions are followed by a fibroplastic reaction This substance is a foreign body and in some will not be tolerated We have left it *in situ* as long as eight years without deleterious effects other than the irritation and adhesion One other patient, however, responded so violently to polythene around the thoracic aorta that it had to be removed within a few days

(*e*) VEIN TRANSPLANT FROM THE AORTA TO THE CORONARY SINUS — Beck[1,4] suggested attaching a vein transplant from the aorta to the coronary sinus It is a formidable procedure By this measure, arterial blood is directed into the coronary sinus from the aorta by the use of a free vein graft The amount of flow must be restricted by keeping the stoma approximately 3 5 mm in diameter The coronary sinus must be open to prevent the pressure in the venous tree from approaching the aortic level Such excessive pressure may cause cardiac failure or myocardial hemorrhage Thrombosis of the grafts is a possibility This method experimentally provides an increased circulation to the heart In the experimental animal, a secondary partial occlusion of the coronary sinus to 3 mm protects dogs' hearts from occlusion of the anterior descending branch of the left coronary artery[17 18]

In Beck's most recent work, the operation (on animals) is done in two stages In the first stage a free jugular vein graft is placed between the aorta and the coronary sinus The stoma at the aortic end was 5 mm in length The pressure in the coronary sinus rose to 18 to 22 mm Hg This pressure did not rupture capillaries but did cause a thickening of the vein

wall In the second stage, a ligature around the coronary sinus near its ostium in the right auricle was tied down to a diameter of 2½ to 3 mm The pressure in the coronary sinus thus rose to 50 to 70 mm Hg Such hearts were then considered protected against coronary artery occlusion Closure of the coronary arteries was then performed experimentally While total occlusion was not followed by recovery, complete occlusion of the descending and the circumflex ramus of the right coronary artery with long term recovery was accomplished Experimentally at least the dogs so treated were better able to stand coronary occlusion in part than control animals [16]

Histologically this arterialization and partial obliteration of the coronary sinus resulted in a fibro-elastic proliferation of the intima The anterior cardiac vein showed little or no change [17]

The future possibilities of this investigative work remain It has not been proven without question that one can revascularize the heart by shunting arterial blood through the coronary sinus That these dogs survive coronary artery occlusion better than control animals appears established It remains for such principles to be applied satisfactorily to the patients with coronary artery disease a sufficient number of times Of the first 12 patients 8 died Twenty three of the last 28 however have survived the operation Of these 23 13 had both stages of the operation The graft was thrombosed in 6 patients and not placed in 2 [17,16] The operation is still in the developmental stage

(*f*) Anastomosis Between Coronary Arteries and the Mammary Artery —Vineberg[18-19] in 1946 demonstrated that one could anastomose the left internal mammary artery to the left coronary circulation by inserting the mammary vessel into the left ventricular myocardium An extra coronary collateral circulation developed in from 50 to 70 per cent of the animals Glenn and his associates[16] showed that this anastomosis was definite This operation has possibilities Further efforts to increase the anastomotic effect by development of arteriovenous fistulas have not proven that this added measure is desirable and its possible ill effects as shown by Holman[22] and Karsner [22a] are well known

(*g*) Other Methods—1 *Other Artery Transplants* —The fact that other arteries not essential to life may be used to carry blood to the coronary vessels has been suggested and is under study

2 *Thrombectomy* —The possibility of a direct removal of a thrombosis has been considered and in some patients may be the ideal treatment Our awareness of cardiac arrest and the immediate thoracotomy necessary in such instances will mean that some patients with coronary occlusion will have their hearts exposed on an erroneous diagnosis of cardiac arrest It may be possible for the surgeon to extract a thrombus in such patients Excision of infarcted areas in the heart wall of experimental animals has been performed successfully The future of these surgical procedures is unlimited

3 *Tube Skin Graft* —Moran[24a] noticed the extreme vascularity attending a tube graft from one identical twin to another The possibility of this type of tube graft as a blood supply to the myocardium is under investigation

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Chapter

# 12

## OTHER OPERATIONS ON THE HEART

*Tumors of the Heart Complete Thyroidectomy Operation for Pulmonary Embolism*

### TUMORS OF THE HEART

Tumors of the pericardium will be discussed in the chapter on pericardium The difficulty of their diagnosis carries over to tumors of the heart itself Until recently no one has been able to diagnosis the condition before death Glenn *et al* recently were able to make a diagnosis preoperatively of a myxomatous type of tumor of the heart which was confirmed at the operating table [6,7]

*Primary Tumors*—More than 300 primary heart tumors have been reported [10,11] Of the primary tumors 126 were myxomas and 114 were sarcomas Five sarcomas were diagnosed correctly during life [3,10,15,16,17] The left heart is more frequently involved than the right The incidence is 0 0017 per cent as found in 480 000 autopsies [18] In addition to the myxomas and sarcomas there were cysts lipomas fibromas lymphangiomas and rhabdomyomas as well as combinations of these tumors reported Some tumors undergo malignant change The rhabdomyomas occur in the ventricle Most of the reported myxomas arose from the auricle *The secondary tumors* affect the right side of the heart most often Carcinoma of the lung pleura esophagus or thymus may affect the organ by direct extension Metastatic growths arise from the kidney the thyroid as well as melanosarcoma xanthosarcoma osteosarcoma and chondrosarcoma [12,13]

**Symptoms.**—The most common sign is the sudden onset of fainting attacks [8,9] There may be loss of weight dyspnea fainting attacks and signs similar to those caused by rheumatic heart disease The $x$ ray and skiagraphic shadow irregularities and peculiarities of the polygraphic tracings may give a clue.[3] There may be pericarditis or heart block due to tumor invasion [6] The absence of a history of rheumatic heart disease may be helpful in ruling out heart disease as is the failure of the patient to respond to cardiac medication [1,20] An early sign may be an arterial embolism [4,21]

**Treatment.**—The treatment of the primary tumors by surgical excision is feasible Modern cardiac and thoracic surgeons no longer fear the cardiac approach Since the diagnosis is so difficult the number which can be removed will be few The primary or secondary malignant ones rarely are removable The diagnosis and surgical treatment of the tumor of the heart performed by Glenn is a noteworthy step in this direction [7,8]

Several other operations have been devised for the patient with heart disease These are of limited use

## COMPLETE THYROIDECTOMY FOR HEART DISEASE

Many patients with advanced cardiac lesions responded to no type of medical therapy The relationship between some of the internal secreting glands and the cardiac status has been recognized for many years The hopelessness of trying to improve the patient in cardiac failure who had any degree of hyperthyroidism was known and such patients despite their circulatory status could only be made better by thyroidectomy

Conversely, the possibility that a hypothyroid state might help the cardiac patient even without thyroid overactivity was entertained and such treatment was attempted. The hypothyroid state with a tendency to reduced activity, lowered pulse rate, and a hypotension all seemed to testify that such a state would aid these hopeless patients Therefore, these patients were subjected to complete thyroidectomy even in the presence of a normal functioning gland. The first reports on improvement of a defective cardiac status by complete thyroidectomy was made by Parry in 1825 [13a]

Blumgart, Levine and Berlin[3a] subjected a series of cardiac patients to complete thyroidectomy operations Babcock[2] and the author[14a] likewise gave this procedure a thorough trial

The mechanism of the relief of cardiac pain after total thyroidectomy is questionable The improvement probably was due to reducing the pace of the heart and slowing the activity of the individual and, consequently, his heart by causing myxedema

Total thyroidectomy should be reserved for the following the patient with cardiac disease who has an enlarged thyroid gland of the diffuse or adenomatous type, or the patient with signs and symptoms of thyroid overactivity whether the gland is enlarged or not

The operation, when performed, is not without its dangers Complete thyroidectomy means total thyroidectomy, as a small amount of gland remaining may hypertrophy and nullify the results Great care must be taken to completely eradicate the gland without injury to the recurrent laryngeal nerve or removal of the parathyroid glands [2, 14a]

The complications of such an operation are (1) any complication of a simple thyroidectomy, such as crisis, hemorrhage, pulmonary complications, hematoma with laryngeal pressure, and wound infection, (2) cardiac failure, (3) tetany, (4) injury to the recurrent laryngeal nerve, (5) myxedema (This latter is a condition which invariably results if the operation is complete It can be controlled by the administration of thyroid extract Myxedema, in itself, has cardiac complications and therefore must be controlled ); and (6) obesity

This operation, therefore, has limited application Its use in selected instances, however, will help the course of cardiac therapy.

## TRENDELENBURG OPERATION FOR PULMONARY EMBOLISM

When a large clot enters the pulmonary artery, it shuts off the right-sided heart circulation If this block continues for a few minutes, certainly

over seven minutes death is unavoidable Surgical removal of such a clot, while ideal theoretically is impossible most of the time The trained team and set-up maintained by most hospitals for cardiac arrest may permit more such operations to be performed in the future The diagnosis must be correct as many will be considered cardiac arrests Where the operating room is ready and no delay is required for permission the Trendelenburg operation could be performed

**Operative Technic.**—The operation consists of an incision over the left second to fourth costal cartilage The pectoral muscles rapidly are divided The chest is opened with a rib spreader The pleura is avoided and retracted together with the internal mammary vessels laterally If this requires extra time the pleura is opened The pericardium is opened The pulmonary artery is exposed a tape passed around it at either end and it is then incised The clot is removed with forceps first from the right and then from the left side The pulmonary artery is sutured the sutures passing entirely through the layers of the wall everting these layers so that the intima comes in contact with intima without the foreign body suture between them

This is a heroic operation with high mortality The shortage of time decreases its applicability Mistaking the aorta for the pulmonary artery has been reported by Capel [4a] Nystrom [11a] and Shamberg [14] Other dangers are the slipping of a clamp used while actually suturing the artery and an injury to the pulmonary artery intima with secondary thrombosis Infection which hampered the early operators can be controlled better with our present day chemotherapy Hemorrhage remains the greatest danger Improved operative technic decreases this latter factor Its greatest field for application would be a pulmonary embolism occurring in the operating room The chest may be opened as in cardiac arrest If a suspected cardiac arrest is atypical a search for pulmonary embolism should be made

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Chapter

# 13

## THE PERICARDIUM

*Pericarditis, Pericardial Tumors, Herniation of the Heart Through the Pericardium*

For this discussion, the pericardium is divided into inflammatory lesions of the sac, tumors and herniation of the heart. Wounds of the pericardium are considered under Wounds of the Heart for obvious reasons (see pages 65 to 70)

**Anatomy.**—The pericardium contains the heart and the base of the large vessels arising from it. It is a fibroserous sac. It lies directly below the lower portion of the sternum and the third to the seventh left costal cartilages. It is in contact posteriorly with the bronchi, the esophagus, the thoracic aorta and the posterior mediastinal surface of the lungs. In its anterior portion, the lungs and the pleura separate it from the thoracic wall except for one portion. It is in direct relation to the thoracic wall on the left side near the left lower part of the sternum and the medial end of the fourth and fifth ribs.[14] On either side the pericardium is in relation to the pleura. The phrenic nerves and vessels descend between these sacs.

The inner serous covering of the pericardium consists of flattened mesothelial cells on connective tissue, the latter being attached to the fibrous layer. This inner layer is in contact with the heart at all points normally and exudes a slight serous fluid. It joins with the outer layer where it covers the large vessels. The fibrous layer is a pouch-like bag. Above, at its neck, it is fused with the outer coats of the large vessels. It blends with these vessel coats and is continuous with the deep cervical fascia. Its base joins the central tendon and muscle of the left side of the diaphragm. It is also attached to the sternum by two ligaments. One passes to the manubrium and one extends to the xiphoid process. It is thus adequately anchored to the thorax. At its upper portion, the epicardium forms two tubes, an arterial and a venous one. The aorta and the pulmonary artery are in the former while the four pulmonary veins and the two venae cavae are in the latter or venous mesocardium. This latter is thus an inverted U in shape. The left atrium is between the limbs of the U. The space behind this atrium and between the limbs is known as the oblique sinus. The space between the two tubes is called the transverse sinus and is actually the area which has the aorta and the pulmonary artery in front and the atria behind. (See Fig. 27.)

Thus, most of the right heart is placed anteriorly. The pulmonary veins are short in their pericardial route, which is of anatomic importance.[32] The arterial supply of the pericardium is from the internal mammary and the descending aorta. This may be of therapeutic importance. The nerves are branches from the vagus, phrenic and sympathetic chain.

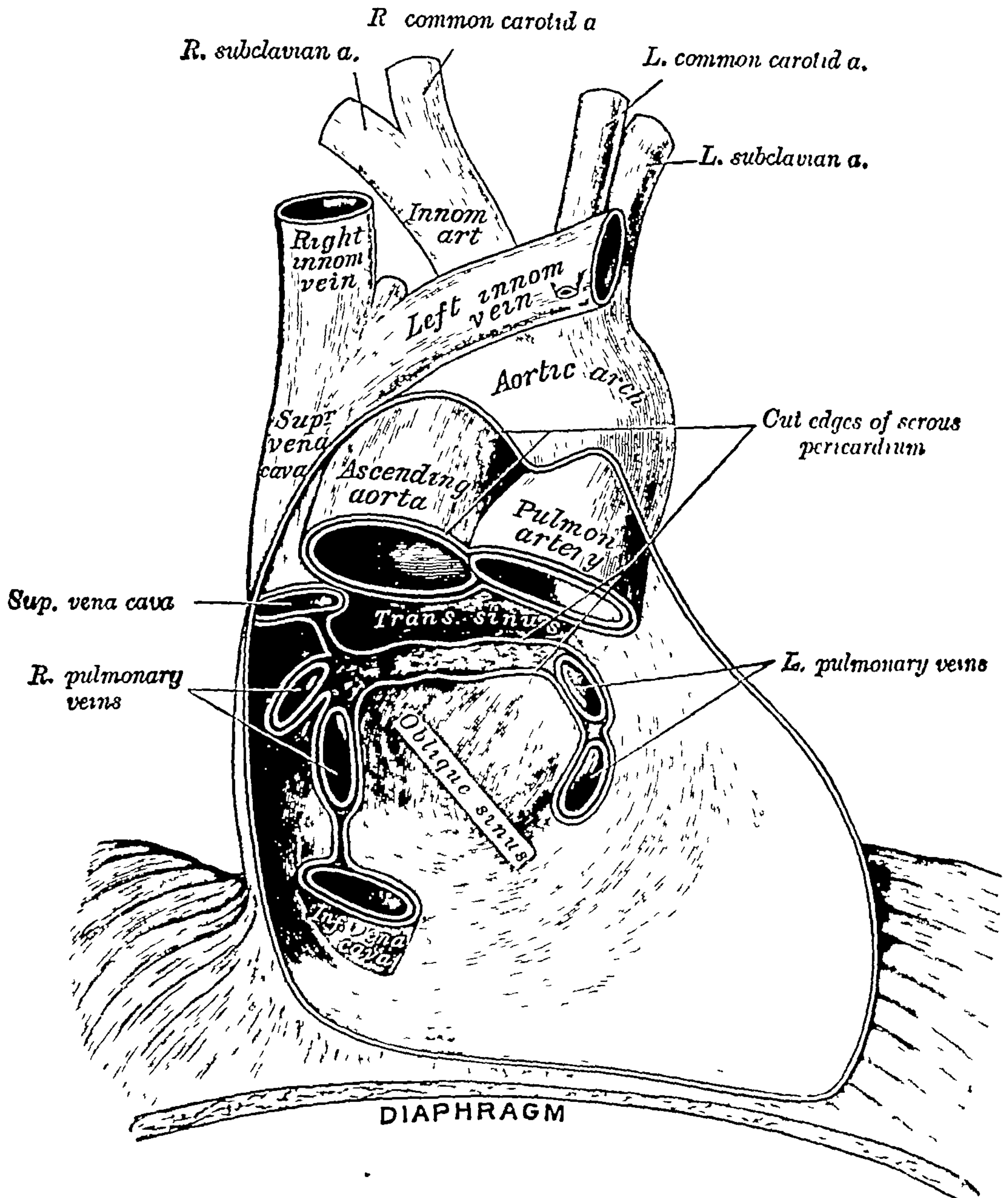



FIG. 27.—Reflections of the pericardium on the great vessels. (Gray's Anatomy.)

## PERICARDITIS

## (INFLAMMATORY LESIONS OF THE PERICARDIUM)

There are four types of acute pericarditis. These are the acute benign, the serofibrinous, that of tuberculous origin and the acute purulent form. These vary as to causative organism, reaction and course. In addition, there is a form of pericarditis characterized by chronic constriction of the heart.

**Acute Benign Pericarditis.**—This subject is of secondary surgical interest as it is only in the complication stage that the surgeon sees the patient. Only recently has this subject become of general interest and of diagnostic significance.

*Etiology.*—The condition is inflammatory in origin and frequently follows an antecedent respiratory infection. The patients usually are young. This lesion is a primary one unlike the acute pericarditis which is secondary to rheumatic fever, uremia, tuberculosis, coronary occlusion or any injury to the sac. The mode of origin of the inflammation is not clear. The existence of the lesion, the significant symptoms, its differentiation from coronary disease and its therapy are of importance.

*Symptoms.*—An abrupt onset of precordial substernal pain initiates the disease. This pain is made worse on breathing and is located to the left of the sternum and may radiate to the shoulder, arm, neck, back or abdomen. The pain is of the coronary type although the latter is not aggravated by movements of the thorax. The respirations are shallow and there may be dyspnea. A pericardial friction rub is of diagnostic aid. The *x* ray shows an increase in the cardiac size. The temperature runs from 100 to 103° F. and there is an increased leukocytosis. The electrocardiograph will help in the diagnosis, especially the reading of the limb tracing. Pericardial effusion may follow.

*Diagnosis.*—The diagnosis is made on the basis of the symptoms. It must be differentiated from myocardial infarction. This is done on the basis of age, the pericardial friction rub, the *x* ray and the electrocardiograph interpretation. Many of these patients are diagnosed as having myocardial infarction and some have been placed on restricted activity without due cause. This lesion also has been confused with an acute intra-abdominal disease. Pain, tenderness, abdominal muscle spasm and a mass in the upper abdomen may lead to a misdiagnosis, particularly when effusion occurs.[22,43]

*Prognosis.*—Most of these patients recover without complications. In others[21,24] an effusion may develop. In some suppuration follows and in other individuals a constrictive pericarditis has resulted. While most of the reported cases have been of a benign nature and complete recovery is usual, the condition can have a more serious outcome. Recently a rapid down hill course with shock and fatal cardiac tamponade has been reported.[22]

*Treatment.*—In many instances the condition is self limited. In some of those following sinus or other respiratory infection, the development of an effusion may require surgical aspiration. If such an aspiration is not sterile a septic origin is in question. Some investigators believe the effusion to be allergic. The possibility that the source of the inflammation is that of a virus has been suggested.[20,27,38,43] The good results with Aureomycin in other virus diseases led to its employment in these lesions.[42] Other antibiotics are used but have not been as effective.

The recurrent nature of the lesion has been noted recently. In one series of 8, the recurrence rate was from 2 to 19 instances.[44] Pericardial calcification occurs in ½ of 1 per cent.[11] Electrocardiographic changes may be residual.

**Serofibrinous Pericarditis.**—In some patients, the development of excessive fibrin in addition to the effusion, occurs early (7 5 per cent of 292 patients) [27] Many with this lesion resolve without sequelae [27] Others form a constrictive pericarditis The surgical correction of this complication is discussed later

**Tuberculous Pericarditis.**—This condition is one of the most serious with which our profession has to deal This disease is insidious, chronic and progressive It results in multiple inflammatory effusions There are fibrous changes with thickening A serious treatment problem is posed which is two-fold. There is an infection which must be treated adequately Also, there are increasing mechanical problems caused by the constriction of the heart and great vessels which interfere with blood flow The obstruction to the venae cavae may be the reason for the pleural and peritoneal effusions that recur so frequently

*Etiology* —The causative organism is the tuberculosis bacillus and, in general, the pericarditis is secondary to advancing and uncontrolled tuberculosis elsewhere in the body In certain patients, the original source may have been healed or "burned out" The continuity of the symptoms in the pericardium in part is due to the healing effort of Nature

*Symptoms* —The symptoms are those of the disease itself with its manifestations in other parts of the body, especially the loss of weight and strength To these are added the specific manifestations of a mechanical interference with circulation Ascites and pleural effusion, in addition to pericardial manifestations, are usual Chills, fever, dyspnea, pericardial friction or effusion are common symptoms

*Diagnosis* —The diagnosis is made on the basis of identification of the disease elsewhere in the body and the local changes in the pericardium and the increasing mechanical interference with the circulation The organism can be recovered from the pericardial tap in a high percentage of patients

*Prognosis* —Prior to the present era, the prognosis was extremely poor. Eighty-three per cent of Harvey's and Whitehill's[20] patients died All of Blalock's and Levy's[9] unoperated patients died and half of those operated also died All three who survived operation were dead within six months Such reports are on patients with advanced pathology however, and probably many did recover who were not in such a serious category as those described Holman[21,22] reported arrest or marked improvement in 5 such patients

GENERAL TREATMENT —Nutrition is of importance as is the replenishment of vitamins In some instances, vitamin deficiency and endocrine imbalance seem to counteract the effect of antibiotics Streptomycin and other drugs should be given in thorough and complete dosages Because of secondary contamination, the other antibiotics and particularly penicillin should be used An occasional patient may improve on medical therapy alone If so, this would occur in the pre-operative preparation stage

Until the recent reports of Holman,[22] early surgical operation was not advocated This was due to the adverse reports of the effect of operation by Blalock,[9] Beck[3] and Churchill [13] Holman's experience with improvement or arrest in 5 patients was the first impressive result with early sur-

ery He stated that streptomycin and the other specific and nonspecific ntibiotics were the primary reason for the modification of his therapeutic onceptions The therapy for the very ill patient is a two-stage procedure n the first stage the sixth left costal cartilage is excised to permit evacuaion of the pericardial fluid In ten to twenty days this is followed by a adical pericardiectomy During both of these procedures, the administraion of streptomycin and the maintenance of nutrition and vitamins begun n the preoperative stage are continued

The combination of streptomycin and para-aminosalicylic acid (PAS) has additive therapeutic effects and delays drug resistance by the organ sm One gram of streptomycin plus 12 grams of PAS are given every two to three days Isoniazid is most effective at the onset of treatment but the organism becomes resistant This drug has been combined with streptomycin A combined streptomycin-PAS-isoniazid therapy with a dosage of 150 to 300 mg of the isoniazid with the usual dose of the other two has worked best according to reports

Surgical Treatment —(a) Early —If progress is being made in the re-absorption of the fluid surgical intervention is not advocated Aspiration to correct dyspnea is always indicated In two of Churchill s patients in which the disease was active at the time of operation the patients both died promptly [14] Aspiration followed by the replacement of air may be of value.

*Technic of Pericardial Paracentesis* —There has been a difference of opinion as to the best technic for needle puncture of the pericardium Romero first aspirated such an effusion by puncturing the sternum in 1819 [28] A recent questionnaire among those engaged in this work showed that most surgeons prefer the anterior apical approach in the fourth or fifth left interspace or subxiphoid approach It is suggested that the tap be made at the outer border of cardiac dullness If a left pulmonary or pleural disease is present or the chest wall is heavy the subxiphoid route is advocated

*Apical Approach* —The patient is placed in a sitting position in bed The skin is prepared and infiltrated with 2 per cent procaine A number 18 needle on a 10 to 50 cc syringe is inserted in the fifth left interspace at the outer border of dullness The needle is directed medially and posteriorly with suction applied to the syringe At approximately 7 to 8½ cm , fluid will be encountered If three attempts are unsuccessful the effort should be abandoned because of the danger to vital structures from the trauma of the puncture.

*Subxiphoid Approach* —For this aspiration the patient is placed with pillows under the back to cause the xiphoid process to protrude. This area is prepared and infiltrated and an 18 gauge spinal type needle is inserted just below and close to the xiphoid process in the mid-line The needle is directed upward keeping the syringe depressed against the abdomen. This permits the needle to follow the sternum until it enters the pericardial cavity It may be necessary to point the needle at a 45° angle to obtain a positive tap The usual distance of penetration for children up to five years of age is 5 cm In adults 7 cm may be required

*Indications for Pericardial Paracentesis* —(1) Cardiac Tamponade Signs: Distant heart sounds, low systolic pressure with paradoxical pulse and venous engorgement

(2) Absence of pericardial pulsations

*Dangers of Pericardial Paracentesis* —Death has resulted This is due usually to laceration of a coronary vessel or the myocardium Ventricular fibrillation may be instituted by the needle The needle may be inserted into a cardiac chamber, the stomach, or other internal organs No one should do a pericardial tap without careful training

*Injection of Antibiotics After Aspiration* —Streptomycin and other drugs (25 to 50 mg in 10 to 20 cc of normal saline) may be injected after aspiration In gross mixed infections especially if the organisms are sensitive to penicillin, 1,000,000 units of penicillin dissolved in 20 cc of saline may be injected

(*b*) Definitive Treatment —Recently, Holman has advocated earlier operation, particularly in those patients who do not appear to be improving [21,22] The moment a diagnosis of cardiac compression is made a cardiac decompression is indicated In the acute stage, he advised that the procedure be performed in two stages In the first step, the sixth left costal cartilage is removed for evacuation of the pericardial fluid Twenty days later, the radical pericardiectomy is performed.

*Technic of Pericardiectomy* —*Incision* —The incision is made to the left of or through the sternum from the second intercostal space to the xiphoid process Sweet[42] advocated a curved incision which ran transversely over the left fourth intercostal interspace from the sternum The fourth and fifth costal cartilages were excised for pericardiotomy For pericardiectomy, the incision begins on the left side at the second costal cartilage and extends down, following a curve to the course of the sixth costal cartilage Recently, an incision has been advocated which extends from one anterior axillary line to the other [24] This incision is made at the fifth interspace with division of the sternum transversely and opening of both pleural cavities The size of the incision depends upon the extent of pericardiectomy necessary A line of cleavage is developed and it is important that this cleavage plane be at a point which permits the heart muscle to bulge between the edges of the incision with each cardiac beat The dissection is made with a scissors in the more adherent points Many areas are free of adhesions In others, the adhesions may be so firm and tight that it may be necessary to leave a small area of calcium on the heart to prevent injuring the muscle itself Some of the areas are so thickened that bone instruments are necessary for their division As much of the calcified pericardium should be removed as is technically possible At the auricular ventricular groove, the deposit may be particularly thick, forming a constricting ring This ring should be divided [42]

During this dissection, one must remember that the coronary vessels are directly under the layer to be removed These vessels must not be injured The cardiac muscle may be particularly soft and easily injured and for this reason it may respond poorly to suture At times, a section of the pericardium must be resutured to an injured area of the heart muscle The amount of resection varies, but in general, the right ventricle, the auricular

ventricular groove the left ventricle and apex and the entire left side of the heart as far back as the inferior pulmonary vein should be freed To control subsequent oozing a catheter should be left or an incision can be made into the left mediastinal pleura If the leakage is minimal this latter measure is preferred An excessive effusion can be removed by tapping If the sternum was split the closure of the wound may be effected by drilling holes in the sternum and reapproximating it with wire The replacement of the sternum in this way permits the ribs to be reapproximated with pericostal sutures The soft tissues are closed in layers thereafter Sweet states that the sternum does not have to be sutured Unless it should cause some cardiac compression it appears technically better to reapproximate this structure and thus regain the normal fixed chest wall

AFTERCARE.—The heart should be protected from the strain caused by a large effusion Prompt aspiration in such instances will relieve cardiac embarrassment Respiratory discomforts should be alleviated by encouraging cough to relieve collections If necessary respiratory fluids may be removed by aspiration or bronchoscopy

**Acute Suppurative Pericarditis.**—*Etiology* —This lesion is rare unless tuberculosis is associated It may be the result of a fulminating inflammatory reaction or it may be secondary to some other inflammatory disease The suppuration which follows tuberculosis often is slow in onset The suppuration after the inflammatory disease most often follows an acute upper respiratory infection which is severe in type In a few no known cause is found Contamination at aspiration time is a rare cause

*Symptoms* —The symptoms of acute suppurative pericarditis are similar to those with acute benign pericarditis but are more severe The onset is sudden with substernal pain made worse on breathing The pain is severe enough to make the possibility of a coronary occlusion a consideration and it must be ruled out in each instance The increase of pain on movement is characteristic of pericarditis. The rapid onset of effusion which later becomes purulent may mask the friction rub at an early date Sometimes the earliest signs are those of tamponade. The febrile reaction is high and may go to 106° F The leukocytosis follows the temperature unless affected by therapeutic drugs

*Diagnosis* —The lesion must be differentiated from coronary occlusion The electrocardiographic tracing helps The increase of pain on movement differs from no change in the pain in the patient with coronary artery disease Patients with this lesion often are in shock and the cause of the shock may be difficult to ascertain X ray findings are typical and help in the differentiation An acute abdominal lesion may be suspected due to the shock, the abdominal muscle spasm and pain At times an epigastric mass is present In questionable cases the aspiration needle may make the diagnosis.

*Prognosis* —The prognosis in suppurative pericarditis of an acute type is serious and guarded The patients die of overwhelming toxemia and tamponade. The latter complication should be prevented by aspiration and/or drainage. If the patient survives the initial attack an adhesive pericarditis develops and this requires later operation

*Treatment* —The therapy requires an early and accurate diagnosis The antibiotic drugs should be administered in enormous doses After aspiration the organism should be cultured and its reaction to the various antibiotics determined The antibiotic drugs can be administered both generally and into the pericardium After drainage is instituted, the sac can be flushed regularly with the correct drug in a saline solution The drainage should not be delayed as tamponade, shock and toxemia will kill the patient without it and at times despite it In suppurative pericarditis due to the tuberculosis organism the points outlined under that disease should be instituted and especially the administration of streptomycin and the other antituberculous drugs The surgical treatment follows that outlined for the tuberculous type Aspiration is followed by drainage, the drain being brought out through a stab wound away from the operative wound This makes the subsequent operation, if needed, simpler technically

**Chronic Constrictive Pericarditis (Chronic Cardiac Compression).**[10,39,40-47] *Incidences and Etiology* —In approximately 2 per cent of all autopsies there is some adherent pericarditis The disease occurs particularly in young men (78 per cent) on the average of thirty years of age In half, the origin is in doubt [19,31] One-sixth of Chambliss's patients had had rheumatic fever, 28 per cent had tuberculosis [12,22]

*Symptoms* —Beck[4,5] described two triads, acute (low arterial and high venous pressure and a quiet heart) and chronic (high venous pressure, ascites and a quiet heart) and these descriptions are still valid The most common symptom is dyspnea Abdominal swelling and peripheral edema coexisted in approximately 75 per cent of the patients Orthopnea, fatigue and the necessity for fluid tappings are usual Over half of the patients had a regular sinus rhythm, but auricular fibrillation (20 per cent) and flutter (7 per cent) may be present

*Pathology* —The pathology varies with the cause The extensive productive changes with tuberculosis has already been described In many instances, the heart and pericardium are a mass of scar tissue The scar may be up to 1 cm in thickness and may be as hard as bone Sometimes the scar invades the heart muscle With extensive disease, the heart may atrophy Histologically, there is an acellular fibrosis with hyalinization Simultaneously, there is fibroplastic proliferation The changes accompanying the specific diseases such as tuberculosis are present in such lesions Pathologic variations in the lungs, liver, spleen and other viscera of a passive hyperemic type consistently occur

*Operation* —In the non-tuberculous patients efforts are made to eliminate active infection The operation consists of the separation of the pericardium from the heart by blunt or, if necessary, sharp dissection At times, a secondary operation is performed to clear the right side of the heart

*Prognosis* —Eighteen per cent of the 61 patients reported by Beck[3] died in the hospital, 6 in the first forty-eight hours and 1 on the table

*Postoperative Results* Recovery occurred in 72 per cent of the 61 patients with improvement The operative mortality was 18 per cent

## PERICARDIAL TUMORS

**Cysts**.—Pericardial cysts are rare. They usually are found on a routine x-ray examination or at autopsy. Ten of Blades's[7] 109 cases of mediastinal tumors were pericardial celomic cysts, but this large incidence probably resulted from routine roentgenograms of Army personnel. Sixteen such cysts had been described up to 1949.[27] Of these only 2 produced sufficient symptoms to bring the patient to a doctor.

*Etiology*.—The cyst is produced by failure of closure of the transverse septum with pleuropericardial or pleuroperitoneal membrane.[41] Blades[7] believes some of these cysts in the past have been called cystic hygromas or lymphangiomas.

*Symptoms*.—Rarely do these conditions cause symptoms. In Pickhardt's[27] case there was a knife-like pain over the precordium. A second patient complained only of an uncomfortable chest sensation.[23]

*Diagnosis*.—Such cysts must be differentiated from bronchiogenic carcinoma, diverticulum of the pericardium or diaphragmatic hernia through the space of Morgagni. Sputum examination, x-ray and bronchoscopy will eliminate carcinoma. X-ray and fluoroscopy should eliminate hernia. A dermoid tumor or teratoma in the mediastinum also is a possibility. In general the diagnosis must be made by x-ray or at operation time.

*Prognosis*.—These lesions are benign. They should be explored surgically to eliminate the tumors that are carcinomas.

*Treatment*.—Surgical excision after exploratory thoracotomy is the treatment of choice. The added risk of such excision after eliminating a carcinoma diagnosis is so slight that it should be performed regularly.

**Tumors of the Pericardium**.—Lesions of the pericardium rarely are diagnosed clinically (25 to 30 per cent). Most tumors of the pericardium are detected by the prosector.

ETIOLOGY.—*Primary tumors of the Pericardium*.[2]—Primary tumors are rare. Lipomas, hemangiomas and fibrous polyps have been reported. Of the 45 reported sarcomas of the heart, 10 arose from the pericardium.

*Secondary Tumors*.—Nearly all such tumors occur by malignant infiltration or metastases. Of 30 reported, 20 were secondary to primary carcinoma of the lung. These tumors make up one-third of all the lesions of the pericardium. None of these tumors was diagnosed clinically. The secondary tumors are similar to those affecting the heart.

SYMPTOMS.—The symptoms are those of the underlying disease. The electrocardiographic changes may indicate extension to the heart. The diminishing of the QRS complex and loss of T wave or slight T-wave inversion is suggestive. Tachycardia not explained by fever is of further diagnostic value.[12]

**Herniation of the Pericardium or the Heart Through the Pericardium**.—A developmental or acquired defect in the thoracic cage can result in a herniation of the pericardium.[8] Severe respiratory and cardiocirculatory difficulties may arise due to the paradoxical motion thus developed.[22,24,25,42] In most congenital instances a failure of sternal fusion causes the defect. The pericardium cannot be closed tightly after operation on the heart

because of tamponade. If the sutures are placed too wide apart however, space for herniation of the heart may be left

*Treatment*—Herniation of Pericardium —The congenital defect can be closed in most cases The pericardial protrusion must be dissected free and replaced Since the patients are children, the thymus usually must be replaced in the thoracic cage By dividing the cartilaginous bands, the sternal bands may be reunited The perichondrium is not interrupted completely laterally to preserve the continuity of the sternum Herniation of Heart.—The defect after operation occurs early and requires immediate surgical intervention Its repair is by more complete suturing of the pericardium

**Secondary Abscess of the Pericardium** —An abscess of the pericardium is rare Such an occurrence was first described by Graves in 1863 [17] Peptic ulcers[30,35] have ruptured directly into the pericardium[1,6,16,36,41,46] and amoebic abscesses have been known to invade this sac The symptoms are not unlike that of myocardial infarction with the additional signs of cardiac tamponade These are a weak heart, venous engorgement of the upper chest and neck, and an increase in all of the symptoms by lowering the head Rarely has the diagnosis been made until death has intervened In a case reported by Meyer,[29] antibiotic therapy for a suspected subphrenic abscess masked the pericardial complication for which surgical intervention might have been attempted

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# SECTION IV

## The Arterial System The Effects of Injury, Disease and Degeneration on the Arteries

## Chapter 14

## OCCLUSIVE ARTERIAL DISEASES

LESIONS which obstruct the arterial supply of various organs and parts of the body cause the most widespread vascular problem in the world today. Two out of every three patients will die of such or associated lesions. These occlusive arterial diseases vary as to cause, age of incidence, degree, site and prognosis. Their symptoms necessarily vary with the site and extent of occlusion. They may depend entirely on the degree of collateral supply available. In many respects however their symptoms are similar and the treatment frequently is the same. The arteries may be occluded traumatically at one site or at a continuous area. They may be subject to disease processes involving a single or multiple site. Metabolic disorders and degeneration also may cause obstruction to arterial blood flow. By occlusive arterial disease is meant those lesions which cause thrombosis within the arterial wall and eventually occlusion such as thromboangiitis obliterans. Some lesion which causes an inflammation in the arterial wall, a subintimal hemorrhage or the production of calcium deposits or plaques may cause an occlusion. Examples of the latter are atherosclerosis, arteriosclerosis or diabetes mellitus. A dysmetabolism of cholesterol with a collection of fat globules plays an important part. All these conditions are characterized by a gradual or acute obliteration of the arterial lumen particularly in the lower extremities. When this occlusion occurs suddenly a calcified plaque is undermined or a thrombus forms. In general the process is a gradual one. With this occlusion, there is ischemia in the part supplied by that artery. This lack of blood supply to the part initiates two types of symptoms. These are first, symptoms of lack of blood supply as shown by claudication, color and temperature changes and at times necrosis or gangrene. Second, due to the lack of adequate circulation an increased susceptibility to infection develops. There are repeated and frequent infections particularly around the digits. Both of these processes may work simultaneously and cause necrosis and gangrene. As these occlusive diseases are similar in their pathology and end result and as this book is concerned particularly with therapy they are grouped together.

## THROMBOANGIITIS OBLITERANS

**Etiology.**—The cause of thromboangiitis obliterans is unknown. Many coincidental changes which occur in patients with this lesion are known. These make the disease a pathologic entity.

The syndrome was loosely described by the Germans under the name of Spontangangran. The first pathological study was made by von Winiwarter[91] in 1879.

Thirty cases of thromboangiitis obliterans were reported by Buerger,[6] who stated that it was a frequent, although not exclusive, disease of the Polish and Russian Jews. More recent work demonstrated that it occurs in all races. Its incidence is highest in those between twenty and forty years of age.

The patients are usually young (under forty years) and are often of the Hebrew, Japanese, or colored race. More often males (99 per cent) are affected, and in 95 per cent of the cases the lower extremities are involved.

Thrombangiitis obliterans is characterized by attacks of ischemia, pain and numbness. On dependency, there is a bright red blush of the toes. Infected ingrown toenails, blisters, corns, etc., are usual. These lesions heal poorly, progress and frequently result in the loss of the involved part.

Most of the patients are heavy smokers. Tobacco appears to be a proven initiator or causative factor. In those who have the underlying disease or a tendency to it, continued smoking will precipitate an attack, causing spasms in the collateral vessels and progression of the disease. If smoking is continued, other treatment fails.

The part that nicotine plays in the production of thromboangiitis obliterans is not entirely clear, but nicotine may initiate the lesions in some patients. Some investigators[61] believe that the disease is produced by the use of tobacco in those individuals who are sensitive to it.

Nicotine plays a part in the onset of spasm. The fact that it may cause occlusion other than by spasm has been proven clinically. The investigations of Wright,[82] Short and Johnson,[32] Maddock, and Coller,[16] Barker,[2] and, more recently, Weinroth and Herzstein[78] emphasize this point.

The occasional non-smoker who develops this disease rules out tobacco as the sole factor. This syndrome has been reported in an order of non-smoking monks in the Himalayas.

Theis and Freeland[71–75] found that tobacco smoking was accompanied by a greater reduction in the oxygenation of the arterial blood in patients with thromboangiitis obliterans than in normal individuals.

**Symptoms.**—One of the earliest symptoms is *claudication*, which tends to progress. There is *pain in the calf or foot or thigh* on walking—at first mild, but later most severe. This pain is intermittent and is always increased by effort and by smoking. There are sensory disturbances, in which *coldness*, *paresthesia* and *anesthesia* are distinctive. On elevation, the foot is pale, on dependency, a marked redness occurs. This *rubor-pallor* (R-P) sign is present consistently. The foot becomes cold. The capillary and the venous filling times are delayed.

The disease often is brought to the patient's attention by a minor cut, abrasion, a blister or a corn which becomes infected and does not heal.

At times minor surgery on such a lesion results in gangrene The ulcerations are usually in the terminal digit and are 'mouse-eaten' in appearance The lesion may progress from an ulcer a part of which is sloughing while another part is epithelializing to a necrotizing area involving one or all of the toes or the whole foot The symptoms depend on the location of the lesion its extent and whether infection is present

Associated thrombitis (thrombophlebitis) occurs in one-third of the patients. In such patients the original arterial defect at first may be masked The doctor always should palpate the distal foot artery in the examination of a patient with thrombitis

The description of the disease by Buerger[4] has withstood the test of time and is quoted below

We usually find it occurring in young adults between the ages of twenty and thirty five or forty years and it is because the gangrenous process may begin at an early age that the names presenile and juvenile gangrene have been employed In one class of cases there are rather characteristic attacks of ischemia The patients complain of indefinite pains in the foot in the calf of the leg or in the toes and particularly of a sense of numbness or coldness whenever the weather is unfavorable Upon examination we see that one or both feet are markedly blanched almost cadaveric in appearance cold to the touch and that neither the dorsalis pedis nor the posterior tibial artery pulsates. When the foot becomes warm some color gradually returns. Some patients complain of rheumatic pains in the leg others are able to walk but a short distance before the advent of paroxysmal shooting cramp-like pains in the calf of the leg makes it imperative for them to stop short in their walk. Some of these cases give the typical symptoms of intermittent claudication After months—or in some cases—even years have elapsed trophic disturbances make their appearance It is at this stage that another rather unique symptom makes its appearance one which gives the foot the appearance typical of erythermalgia In the dependent position a bright red blush of the toes in the anterior part of the foot comes on rather rapidly extending in some cases to the ankle or slightly above Soon a blister hemorrhagic bleb or ulcer develops near the tip of one of the toes usually on the big toe, frequently under the nail and when this condition ensues the local pain becomes intense. Such trophic disturbances may at times make little progress and last for months sometimes however the skin in the neighborhood shows cyanotic discoloration and dry gangrene of the whole toe is an early issue '

**Pathology** —The soft clots which form in the lumen of the artery later become firmer and may occlude the entire vessel When one dissects an amputated leg or the arteries of an autopsied patient he finds often that the occlusion is localized These clots usually are slow in development and may recanalize in one or several places This recanalization leads to the remissions of the disease. In cross section the many openings in the vessel give an appearance of a Gatling gun

With the closure of a large vessel there is usually some degree of spasm in the collateral vessels. This is accentuated by overuse or taking of vasoconstricting drugs such as nicotine or ergot or the local injection of procaine or other anesthetics

The occlusion occurs in a large proportion of blood vessels, with the thrombosis extending distally but not proximally The occlusion will involve the anterior tibial, the dorsalis pedis, and posterior tibial arteries, without necessarily involving the peroneal artery. There may be no intimal thickening In older patients, there will be an accompanying arteriosclerotic change With organization of the thrombus, there will be intimal thickening

**Diagnosis.**—While this condition is primarily seen in the lower extremities, it also involves the upper extremities (5 per cent) Other organs of the body may be involved, at times similar to the general involvement seen in arteriosclerosis Visceral lesions are present in less than one-half per cent

In making a diagnosis, other obliterative arterial lesions must be ruled out The usual factors for making the diagnosis are (1) arterial insufficiency, (2) male sex, (3) forty years of age or less; (4) smokers, (5) juvenile or gangrene at a relatively early age Factors contributing to the diagnosis are (6) a migrating thrombitis, (7) remissions of symptoms and signs, (8) no arteriosclerosis elsewhere in the body, (9) the lower extremities

When the process is advanced, the dorsalis pedis and/or posterior tibial pulsations are not felt As the process continues, the popliteal and even the femoral pulsations may disappear The oscillometric readings will be diminished or absent At times, the occlusion is distal to the major vessels, these vessels still being palpable

The characteristic pain is a differential point It is a severe, excruciating pain which the patient finds intolerable It is worse when the part is used but persists at night With dependency of the extremity and/or squeezing of the foot, some edema develops which causes a local pressure and thus relieves some of the pain, acting similarly to the infiltration of a local anesthetic For this reason, the patient reports that dependency gives relief of pain

From the pain and lack of sleep, the patient becomes nervous, irritable, and usually is described as "high-strung" Such patients have a strained, tense appearance Mild sedation is soon ineffective When seen by the specialist, many of these patients already are addicted to drugs The addiction adds to their pain by increasing their nervousness and irritability when they are not under the influence of the drug.

If arteriosclerosis is likely to be present, a roentgen ray, or even a biopsy, may be necessary to establish the diagnosis

*Arteriograms*—An arteriogram made with a radiopaque dye will show the site of the block or blocks Collateral circulation around such blocks likewise can be demonstrated. The possibility of the dye causing a spastic occlusion of these collateral vessels must be considered Its routine use, therefore, as a diagnostic procedure is limited to those patients in whom the possibility of resecting or by-passing the block exists

**Treatment.**—A PROPHYLACTIC MEASURES —The conservative management of all occlusive arterial diseases is similar Since the disease is progressive, one must know and practice, with the cooperation of the patient, the necessary conservative measures to limit the lesions.

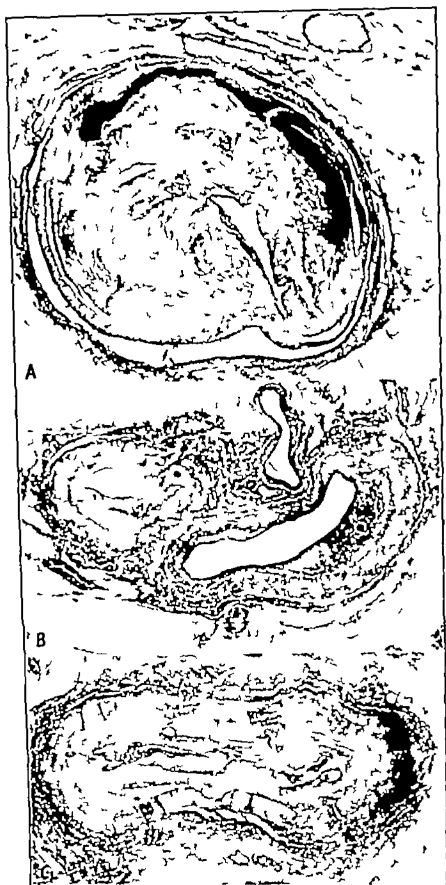



FIG 28 —Pathology A in thromboangiitis obliterans organized and recent thrombus shown.

B Later fibrotic changes in medium sized arteries with partial recanalization

C Late changes in thromboangiitis obliterans. Vessel replaced by organized thrombus which has been fibrosed

These changes vary microscopically but grossly are not unlike those occurring in arteriosclerosis.

The following conservative measures apply to all types of occlusive arterial disease

1 *Elimination of Physical Vasoconstriction* —Constricting bandages, garters, tourniquets, corsets, casts, etc, must be so applied as to avoid or remove constriction to the blood vessels

2 *Elimination of Chemical Vasoconstriction* —The chemicals particularly are such drugs as nicotine, adrenalin, ergot, whose vasospastic action is well recognized

(*a*) *Cessation of Smoking* —In all patients with arterial occlusive diseases, improvement from any treatment cannot be expected unless the

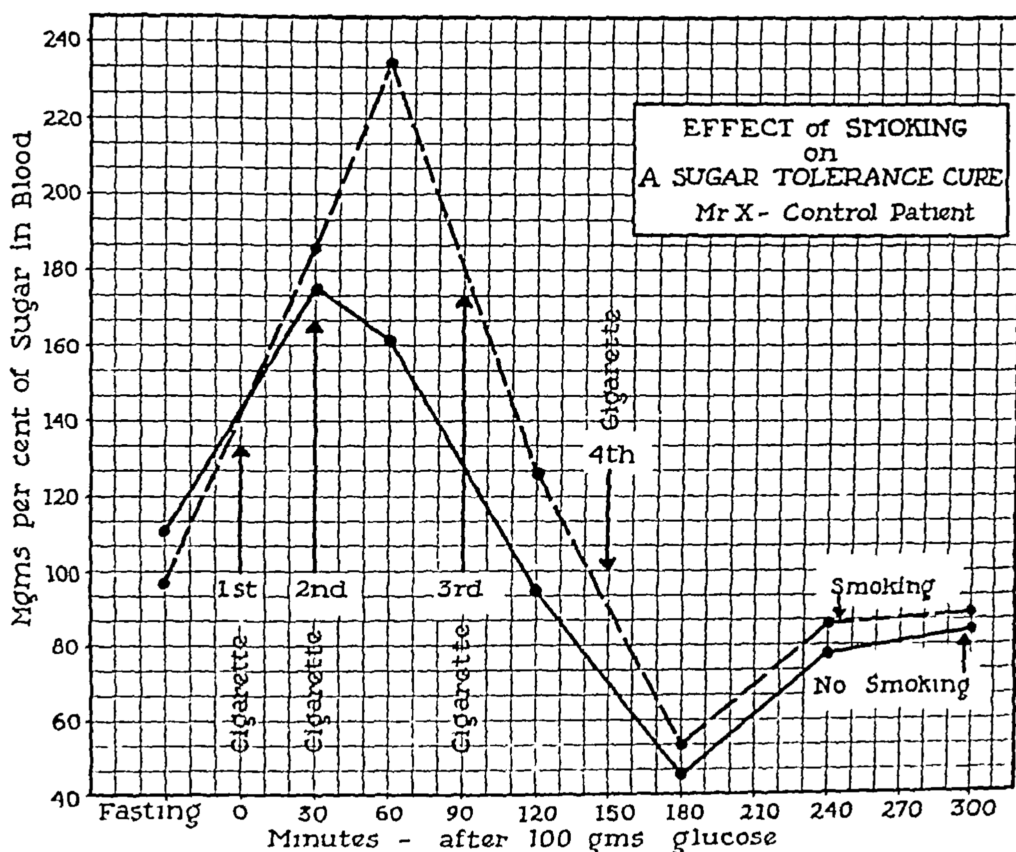



FIG. 29 —Graft depicting the increase in blood sugar on the glucose tolerance curve following the smoking of cigarettes The effect is even more striking in those with arterial disease (Courtesy, Dr F V Theis, Chicago)

patient stops smoking So important is this factor that some vascular clinics refuse to institute therapy if the patient refuses to cease smoking Such a practice tends to make the patient dishonest and keeps the problem hidden rather than out in the open where it can be discussed and perhaps controlled Psychiatric help may be needed

The spasm-inducing effect of nicotine on small blood vessels is well-known It can be demonstrated in laboratory animals and has been observed clinically With the patient's major blood vessels occluded and his collateral vessels kept in spasm, the loss of the extremity is unavoidable. Nicotine itself may cause an arterial occlusion in the patient who is

susceptible to the drug Wright,[illegible] Johnson and Short,[illegible] Silbert[illegible] and Weinroth and Herzstein[74] are all on record as stating that this is true. It may be that if the tendency to occlusion or sclerosis is inherent or acquired through some antipathy, nicotine may precipitate that tendency.

The nicotine habit is one of the most serious problems with which we are faced. The smoking habit is formed early—in high school and sometimes before. Through aggressive national advertising via billboards, newspapers, magazines, radio, and particularly television, the merits of tobacco products are so extolled that it becomes fashionable to smoke, thus producing nicotine addicts of both sexes at a surprisingly early age.

Nicotine may prove to be our national menace similar to the Chinese opium habit. With the knowledge that two out of every three people after the age of forty will have some lesion of the heart or blood vessels with which smoking is incompatible, there is much support for such a premise. When one observes men and women, otherwise strong willed, who lose an extremity merely because they are unable to control their tobacco addiction, such a concept is strengthened further. During war times service men and women are supplied with free cigarettes and "smokers" are part of their social life. Two-thirds of battle wounds involve the extremities. It was not unusual to see wounded men brought in, often without needed plasma or surgical dressings but rarely without a cigarette hanging from the lips. Since many such wounds involve the blood supply to the part, nicotine was adding the spasm factor and contributed many times to an amputation. Since the human is a creature of habit, it will be necessary probably to replace the smoking with some other practice. The search for some substitute for nicotine eventually will be successful. Roth's[84] work has contributed to the vast evidence that has accumulated to support the difficulty of breaking the smoking habit.

Nicotine also is harmful to the diabetic because it will release sugar into the blood stream. Lundberg and Lundberg[44] demonstrated an increase of 50 per cent in the blood sugar of a diabetic after the patient had smoked two cigarettes. Nicotine produces an adrenalin-like reaction which releases glucose into the circulatory system.

(*b*) *Other Drugs.*—Adrenalin is a strong rapid-acting vasoconstrictor for peripheral vessels. Its inclusion in procaine or for other therapeutic measures may act adversely on the circulation. Such drugs as epinephrine, neosynephrine, and the other substances utilized for the shrinking of mucous membranes may have a deleterious effect. Ergot, which has routine use in obstetrical and neurological fields, can cause serious vascular spasm in those with diseased vessels. If one infiltrates soft tissues near diseased vessels, as in local anesthesia, constriction of the vessel may occur. This same point applies to the technical errors of intravenous fluid injection or hypodermoclysis.

3. *Elimination of Trauma or Infection.*—Trauma or infection may lead to skin breaks, necrosis, and even gangrene. This point cannot be overemphasized. The patient should be instructed in hygiene. A neglected corn or callous, harmless to the normal individual, may be the beginning site for a major arterial occlusion in a patient with thromboangiitis obliterans. A minor trauma is not unusual in the history of the onset of gangrene. The

pressure of an ill-fitting shoe, a short stocking, the stubbing of a toe, the burning of the part with a hot water bag, or the use of some chemical to remove a corn, or a fungicide may initiate gangrene A nail scratch, a blister, or someone stepping on the foot, in these patients, may cause a wound which will not heal

Antiseptics are tissue irritants Strong chemicals used in the treatment of athlete's foot (*i e*, salicylic or benzoic acid) may cause a permanent unhealed wound in the patient with an obliterative arterial disease

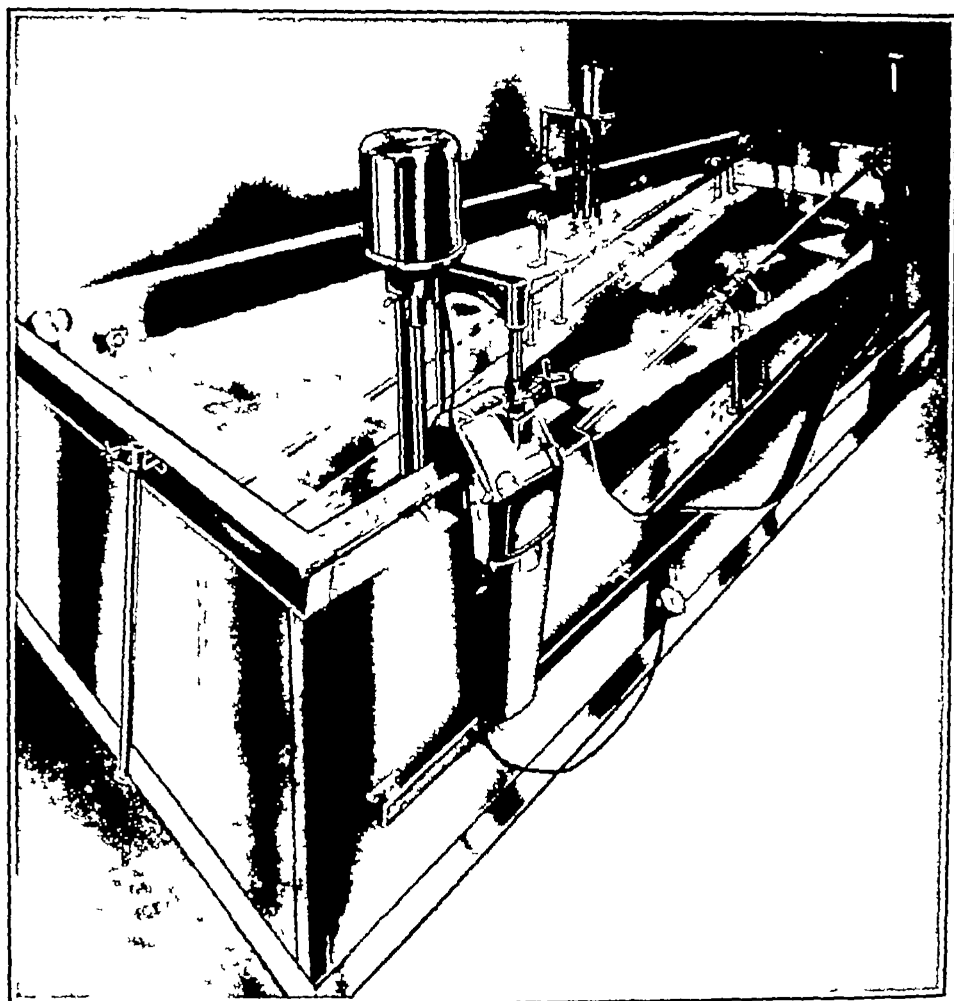


FIG 30 —Rehabilitation tank Courtesy Dr S Sverdlik, Department of Rehabilitation, St Vincent's Hospital Such a tank, combined with whirlpool, is of value in removal of purulent and slough material without trauma A sulfonated detergent (pHisoHex) is added to the water Adjustable platform permits exercises

4 *Collateral Circulation Stimulation* —Whatever collateral circulation remains when the main blood vessel closes should be stimulated and developed in every possible way. Some of the more effective measures are detailed

B MEDICAL MEASURES —Each of the following points have been time-tested in the Cardiovascular Surgical Clinic of St Vincent's Hospital The procedures may be modified according to the individual needs of the patient

(a) *Bed Rest* —In an acute attack bed rest is most important and must be continued until the emergency phase has passed Bed rest reduces the demand for blood by the extremity There should be active and passive motion of the limbs *in bed* to reduce arterial and venous stasis, but weight bearing is avoided

(b) *Exercise* —As the acute phase passes use of the leg below the fatigue stage is one of the most effective methods to stimulate the collateral vessels Walking should be kept within the ability of the vessels to supply blood to the extremity Judicious use of the involved extremity will keep active the circulation that is present and thus decrease the tendency to thrombosis. A helpful exercise is swimming in a warm water pool especially in salt water The buoyancy of the water reduces the need for strenuous activity Resting on pneumatic rafts or tubes and mild movement or paddling with the limbs also is helpful Tanks and whirlpools are adaptable to hospital use (see Figure 30) [70] A further use is to clean drainage and debris from a local wound To the warm water can be added a sulfonated detergent with Hexachlorophene such as pHisoHex Violent exercise motion sudden flexion or extension of the joints may loosen and break off a plaque or sclerotic area

(c) *Foot Care Hygiene Shoes Arches* —Proper care of the feet is an important habit to inculcate early in life for the eventual health of one s legs may depend upon rigid observance of these simple hygienic measures The feet must be assumed always to be unclean Stockings and shoes and walking contribute to constant perspiration Patients with obliterative arterial diseases should wash their feet several times a day with a bland non irritating soap solution and dry them carefully with a clean soft towel to be used only for the feet A sulfonated soap detergent containing Hexa chlorophene (*i e* pHisoHex) is suggested Care should be taken to avoid skin breaks by rubbing A mild lanolin cream may be used to overcome excessive dryness or scaling The control of the fungus infection which all these patients develop will be mentioned later At the Cardiovascular Surgical Clinic of the St Vincent s Hospital patients are told to wash their feet more carefully and more often than they would their faces The care of the nails is stressed Nails should be cut by some trained person other than the patient They should be trimmed with sterile scissors in a good light after a careful soap bath The nails should always be cut straight across to avoid ingrowing of the nail or cutting of the skin After the cutting the feet should be washed again Cramped shoes and stockings play their part in the development of corns callouses and pressure points. Patients with obliterative arterial diseases must wear sensible shoes which give the foot ample room and eliminate pressure. Shoe designers emphasizing pointed constricting footwear contribute to the already complicated problem

Where there are serious arch defects these must be corrected by properly fitted arches made from casts for the individual patient by trained arch makers. The exercises usually employed to strenghten the arch are too strenuous for the patient with a vascular lesion New straight last shoes are needed as the corrective arch changes the shape of the foot and the old shoes will cause pressure. A warm tub bath daily in water over the hip

level (88° to 92° F ) is an excellent therapeutic measure Ointments which destroy tissue should not be used Irritating antiseptics should be avoided Corns and callouses represent pressure points and are self-limited with the removal of the friction site A skilled podiatrist who understands the fundamental vascular problem is invaluable to a clinic or to the vascular surgeon

(*d*) *Temperature.*—Most vascular lesions are best treated at room temperatures, avoiding extremes of hot or cold *Warmth*—While room temperature works well in some patients, the external warmth supplied by a light cage appears to be beneficial and aids in reducing spasm If warmth is used, 82° to 92° F is a safe range of temperature It is important that each cradle be large and contain a thermometer, and the temperature must be controlled

Thermostatically controlled cradles, such as the Vasculator (Fig 31), are available

(*e*) *Reflex Heat* —The benefits of such therapy are best obtained by applying a hot water bottle or electric pad on the abdomen or by immersing the hands in warm water The skin temperature of the feet in a normal individual can be raised from 10° to 15° F in this way

This is the basis of the *Landis test* to determine the individual's ability to respond reflexly and thus to judge how much vascular spasm is present

(*f*) *Refrigeration* —The disproportion between the oxygen demand of the tissue and the oxygen supply available when the main blood vessels close has been a matter of considerable study Maintaining the temperature of the affected extremity is one way to increase the oxygen supply. Lately, following considerable experimental work by Fay,[19] Crossman and Allen,[7] Freeman,[20] and other investigators, an effort has been made to produce the same result by decreasing the oxygen demand of the extremity by cooling it locally

Murray, Simpson, and Watters[50] reported on the value of combining dependency and local refrigeration with sympathetic block in patients with an acute occlusion in whom other forms of therapy were contraindicated Limbs in this stage have been observed to recover with only nerve blocks and at times without any therapy

In the majority of patients, efforts to supply more blood and reduce spasm of the extremity are better treatment than attempting to decrease the oxygen demand. Wright[81] and Duryee[83] agree with this view

When the extremity appears lost, then the part should be refrigerated The use of lower temperatures to treat patients whose arterial circulation definitely is inadequate or appears to be hopelessly lost is supported by experimental studies on animals If the traumatized extremities of the experimental animals were iced before tourniquets were applied, the mortality dropped 50 per cent [90a] This indicated that in ischemic extremities the application of ice reduces the mortality rate

When occlusion is advanced and further efforts to increase the blood supply appear futile, in certain instances improvement has followed the use of refrigeration The value of refrigeration will be further discussed in the section on amputation (see pages 239 to 240). Like all other forms of treatment, ice therapy should be adapted to the individual needs of the

patient The refrigeration blanket or more recently the ice tent is a satisfactory cooling medium when available

*Refrigeration With Hypothermia During Operation* —It has been observed that hypothermia during an operation reduces the blood loss, permits longer periods of anoxemia to the limbs and brain without deleterious results and decreases the amount of necessary anesthesia This modality has a place in selected instances where the risk of the hypothermia is less than that to be encountered by anoxemia or blood loss This problem is discussed on pages 34 to 36 The therapeutic possibilities of refrigeration both in the operating room and elsewhere have not been exhausted

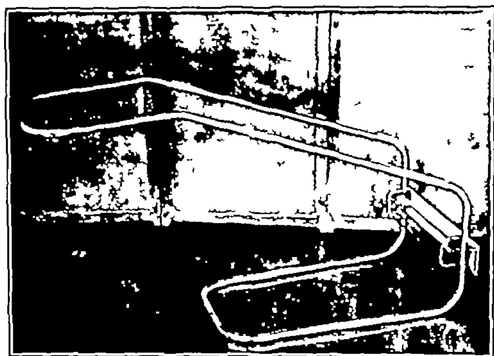


Fig 31 —Bed cradle with side pieces prevents injury of the extremities Cradle keeps clothing off of extremities Thermostatically controlled heat can be added (Valverde) as can ice bags.

(*g*) *Postural Changes* —A change in the posture takes advantage of changes in gravity which are caused by raising and lowering of the part Buerger originally suggested postural exercises based on the fact that the blood would drain from the foot on elevation and would fill on dependency The time necessary for this to occur varies greatly in different individuals and in different stages of the disease The time of elevation and dependency should be varied depending on the length of time it takes for these color changes to occur It will be observed that in one person the blood will drain from the part in thirty seconds on elevation while in another it may take from three to four minutes Buerger's original exercise of two minutes elevation two minutes pronation and two minutes dependency therefore should be modified as required for the individual under treatment

The oscillating bed (Saunders) provides continuous exercise of the Buerger type It is non traumatizing Blood rushes into the feet when the foot

of the bed is lowered and the extremity blanches when it swings upward This is principally a venous change, but some arterial improvement may follow this venous flow In this way, some blood may drain through the capillary bed

As most patients feel better when the leg is dependent, this period may be extended, but only until rubor is present The period of elevation should be continued only until the pallor appears In the acute stage, raising the head of the bed helps the circulation by the action of gravity Elevation of the extremity in the acute occlusion stage, as is so often done, is contraindicated This stems from our early tribal life when an injured part was raised to get it out of the dirt

(*h*) *Foot Soaks and Fungus Care*—Soaking the feet in a weak salt solution at a temperature of 88° to 92° F for thirty to sixty minutes, two

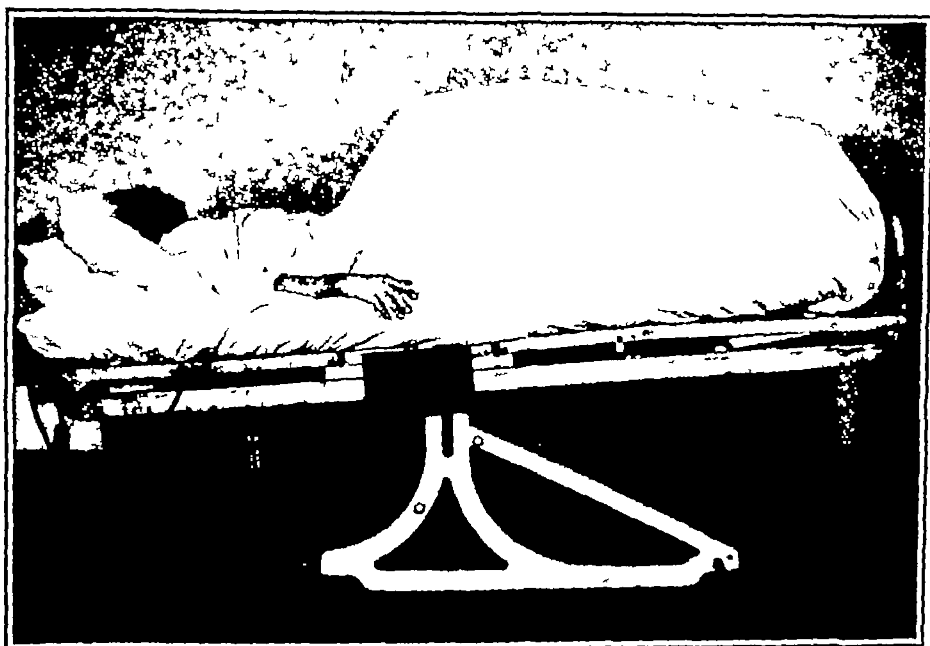


FIG 32 —The Saunders Oscillating Bed This is a passive vascular exerciser of an atraumatic nature

or more times daily, is helpful not only as a local vasodilator but also to soften and aid drainage of any nail edge infections

Since most patients with an obliterative arterial disease have an associated fungus infection, a foot soak in a 1 15,000 to 1 25,000 potassium permanganate solution for twenty minutes, at two- to four-day intervals, will keep the infection under control and prevent the skin breaks so dangerous from the secondary infection standpoint

This fungicide is mild Within the last fifteen years only five patients have been seen who were allergic to the solution This soak helps control the itching

(*i*) *Whirlpool Bath, with Postural Changes*—Many patients with open lesions have drainage which is difficult to remove without traumatizing the tissue The whirlpool will aid painlessly in this important work We have

combined this atraumatic drainage with the beneficial effects of postural changes contained in the use of the oscillating bed. With the aid of the Rehabilitation Clinic at St Vincent's Hospital we have had constructed a tank to fit on a Saunders-type bed. The fluid enters and leaves the chamber in which the foot is emerged with the elevation and descent of the bed. To the warm water is added the detergent soap with Hexachlorophene (3 per cent) (pHisoHex). Tissue cultures have shown that the organism count of the washing fluid is reduced after the use of this agent, thus demonstrating the value of the mild mechanical wash, the postural change and the detergent action of the soap with Hexachlorophene.

(j) *Removal of Focal Infections.*—The part focal infections play in occlusive arterial diseases has not been stressed sufficiently. The painful arterial spasms which attend occlusive arterial lesions can be decreased or eliminated by removal of the devitalized defective teeth or other focal sites of infection.

The exact mechanism by which a focal infection interferes with the arterial supply to the extremities is not known. It has been shown that as much as 60 pounds of pressure may be exerted when a person forcibly bites on his teeth. The apical abscesses may be traumatized and infection liberated into the general vascular system. Such action by a reflex vasoconstriction may cause spasm or occlusion in already diseased arteries.

Infected or devitalized teeth and root abscesses should be promptly eliminated.

(k) *Tissue Extracts.*—Certain tissue extracts, principally those derived from the pancreas, seem to alleviate the symptom of claudication in the arteriosclerotic patient. They act by interrupting the vasoconstriction that follows adrenalin administration.

Other tissue extracts are reportedly helpful.[50] Such tissue extracts are not cumulative. While not of proven value, the extracts are not injurious.

(l) *Vasodilators.*—Vasodilators, such as papaverine hydrochloride, eupavin, salicylates or whiskey may aid in overcoming some of the acute spasm.

Of the available vasodilators, papaverine hydrochloride in large doses has some effect. It belongs to the isoquinoline group of opium alkaloids. It acts on the smooth muscle cells of the blood vessel walls.[1]

(m) *Interruption of the Sympathetics.*—Chemical interruption of the sympathetics is helpful in the acute episode with the collateral circulation in spasm. This can best be performed by a paravertebral sympathetic nerve block with novocain 1 to 2 per cent solution or in oil with benzocaine and benzyl alcohol or with an anesthetic in oil for a more prolonged effect. Where sympathetic interruption effect is desired for longer than that supplied by a local block, a continuous drip can be instituted. A polythene tubing is inserted through the needle and the needle withdrawn. Procaine can be dripped in the polythene tube. The possible effect of accumulation must be remembered. Procaine solution should be diluted 0.1 to 0.2 per cent solution. The total dosage should be dependent upon the patient's age and status.

Intravenous ether or intravenous novocain provides a temporary sympathetic effect but have the disadvantage that they are generalized in nature

When the sympathetic nerves are interrupted and more blood enters the desympathized limb, blood is drained from other parts of the body.[11] If the left lumbar sympathetic ganglia are anesthetized, the left leg becomes warm, while the right leg is cooler. The available blood volume in the body is a definite quantity; if more blood is required for one part, some other part or parts of the body will be deprived of their blood supply.

The borrowing and lending system of blood supply acts like the Federal Reserve Bank which helps a member branch bank when its depositors

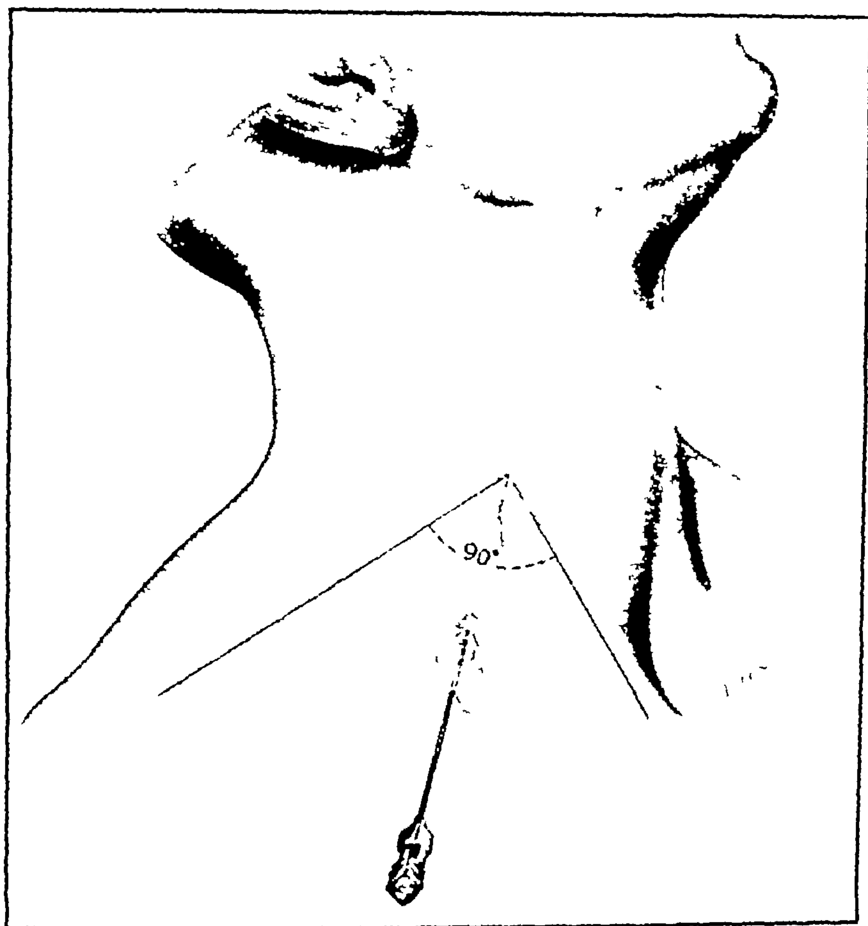



FIG 33 —Anterior technic for sympathetic nerve block of the upper extremity. The stellate ganglia is anesthetized and by infiltration anesthetizes sufficient sympathetics to give the desired effect in the upper extremities. (Pratt, Surg Clin N A, 1943, courtesy of W B Saunders Co)

suddenly withdraw their money. This explains the efficacy of a block of the sympathetic ganglia of the extremity in contradistinction to some generally acting drug which draws blood from everywhere and tries to supply blood to all parts of the body.

A combination of histidine monohydrochloride and vitamin C (sodium ascorbate) has been advocated as a vasodilator.[25] In our experience as well as that of others, no startling therapeutic effect has followed its use. The cholines, such as "Mecholyl" (acetyl-betamethylcholine), when used locally, may be helpful in aiding local vasodilatation.

*Sympatholytic and Adrenolytic Drugs.*—Tetraethylammonium chloride gives a transient sympathectomy effect. It has a temporary therapeutic reaction. Severe hypotension may follow and deaths have been reported after its use. Priscoline* and Dibenamine** also cause sympatholytic or adrenolytic action of a somewhat unpredictable nature. These substances are discussed completely in the chapter on Interruption of the Sympathetics, pages 491 and 492.

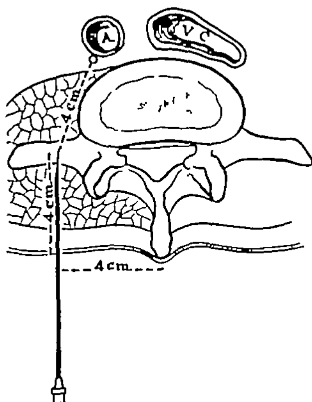



FIG. 31.—Lumbar sympathetic nerve block, author's technic. The patient lies on abdomen (or side) and the lumbar curve is corrected by a pillow. A point 4 cm. opposite each intraspinous space of L1 to L4 is selected. A needle is introduced slightly angling toward the body of the vertebrae for a distance of 8 cm. Using this intraspinous area avoids striking the process which causes pain and movement. The needle is aspirated to be sure the needle is not in a vein or in the dura prior to the injection of the solution.

(*n*) *Anticoagulant Therapy.*—The use of anticoagulants in the acute and the chronic arterial occlusion is past the experimental state and the results are promising. (See chapter on Antithrombotic Substances, p. 651.)

In the acute occlusion heparin should be given at once and until the prothrombin time has been lengthened by the oral anticoagulants.[13] Heparin can be given intermittently with an initial injection of 35 to 50 mg. of sodium heparin intravenously followed by 50 mg. subcutaneously (deep) in one hour. An average dosage of 30 to 70 mg. subcutaneously every three hours can be checked with the coagulation time of the patient to keep the coagulation level between twenty and thirty minutes. Heparin

*Priscoline—2-benzyl-4,5-imidazoline hydrochloride.
**Dibenamine—dibenzyl beta-chlorethyl amine hydrochloride.

has been used in large doses. Two hundred mg. subcutaneously will change the coagulation time for twenty-four hours. One must watch for hemorrhage at the end of three hours, but such a possibility can be reduced by pressure bandage and ice bags to prolong the absorption time.

One of the oral anticoagulants such as Dicumarol or Tromexan or others should be started at once. (See chapter on Antithrombotic Substances, pages 651 to 666.) Subsequent dosage is determined by the daily prothrombin level, which should be kept at 2 to 3 times the normal. When the effect of the oral anticoagulant selected is apparent, the heparin may be discontinued. Several other drugs with similar anticoagulant properties have been developed.

(*o*) *Drugs that Reduce Blood Viscosity.*—A hypertonic saline solution, as advocated by Steel,[65] Silbert,[59,60] Samuels[56] and other workers, theoretically acts by decreasing the viscosity of the blood and increasing the total circulating media. A few investigators have reported good results with this treatment.

Theis's[71,75] reported good results with sodium tetrathionate ($Na_2S_4O_6H_2O$) and sodium thiosulfate ($Na_2S_2O_35H_2O$) have not been repeated regularly by other researchers. He combines this therapy with Pavex boot treatment.

(*p*) *Suction Pressure Boot or the Pavex Machine.*[27,28]—This boot is a machine with positive and negative pressure which may help in certain cases but in others has caused harm by traumatizing the limb. Special care must be taken to prevent trauma. We have discontinued its use. The theory that one can force arterial blood through the capillary bed mechanically is unproven. Herrman is extending the boot to include the pelvis.

(*q*) *Hyperemia.*—Hyperemia means an excess of blood in any part of the body. When it is due to an increased inflow it is called active. A warm bath or saline soak can increase the circulation locally. Bier induced venous congestion by applying a rubber band. He used this for joint infections and other inflammatory conditions. The employment of such a measure in arterial occlusions could cause serious damage.

The use of some drug or compound to cause local inflammation has been advocated as an aid in increasing the blood supply. Any skin irritant such as mustard will produce such a reaction. Some doctors have claimed that their paste is a "secret formula" and have claimed beneficial effects for their salves which could have been obtained with a warm bath. The use of such irritants cannot be advocated. Many patients have been drained of their meager savings in this way for a treatment they could reproduce readily in their own bathrooms. Warm packs have a definite place in therapy.

(*r*) *Intermittent Venous Occlusion.*—A pressure cuff has not been proven to be of value. The theoretical basis of this form of therapy was to develop a constricting phase to block the venous outflow, and then by rhythmical release of this constriction to hope to suction the arterial blood through the capillary bed of the affected extremity. It was necessary for the State of New York to pass laws that restrict the distribution of such apparatuses by the laity on a rental or sale basis without supervision.

Very few authors still advocate the use of this method.

Because of trauma and equivocal results this method is no longer used in the Cardiovascular Surgical Clinic of the St Vincent's Hospital The report of Smithwick and others confirmed our own opinion that such a measure was ineffective [62]

(*s*) *Typhoid Vaccine* —Typhoid vaccine may be a limited aid in stimulating the collateral vessels My associates and I have been using typhoid vaccine as an adjunct in the treatment of thromboangiitis obliterans for over fifteen years The initial *intravenous* dose of 500 000 *killed* typhoid organisms is increased at three- to seven-day intervals to the tolerance dose



Fig. 35 —Arteriosclerosis in the femoral artery in a child aged 4 Trauma was the precipitating factor Note the ulcerations of the knee and ankle and atrophy of the affected leg X-ray shows sclerotic plaque deposit (Courtesy Capt L Bell M C U S N San Diego Calif )

The desired reaction is an elevation of the body temperature to approximately 101° F for three to four hours It is imperative that the dosage be carefully regulated so that a chill is not produced as spasm follows chills A satisfactory typhoid vaccine may be prepared with a concentration of 100,000 000 *killed Eberthella typhosa* per cubic centimeter There is no U.S P standard potency for this preparation A fresh preparation should be selected The solution should be agitated before injection to make the suspension uniform The vaccine should be prepared for intravenous use only The potency of each new vial of typhoid vaccine to be used should be determined by administering it in small doses to observe its reaction

Special Therapy for Thromboangiitis Obliterans —Since the conservative management of occlusive arterial diseases is similar whether the

cause is thromboangiitis obliterans or one of the sclerotic types of lesions, these diseases have been discussed together. Certain treatments have been advocated as specific for thromboangiitis obliterans. To our knowledge no such specific therapy exists.

*Diet.*—No type of diet has been found to be specific for these patients. Obesity always should be prevented. In the manufacture of the rye bread in eastern Europe, ergot was in the flour. Since this is not encountered in the United States and probably nowhere else with modern baking, the eating of rye bread poses no problem.

*Climate.*—Many doctors instruct their patients to live in a warm climate. This induces economic difficulties in most cases. There is no less incidence of amputation in a warm area than in a cold one provided the fundamental hygienic care is exercised.

*Foreign Protein Therapy.*—Typhoid vaccine has been used for many years. The febrile reaction may increase vasodilatation.

*Hypertonic Saline Solution.*—This problem is discussed under the management of all occlusive diseases. It is the basic treatment in many clinics. Our results were not encouraging.

*Antibiotic Therapy.*—These drugs should be used wherever there is an open lesion. The specific one for the cultured organisms should be selected.

*Vitamins.*—Vitamins should be supplied to those who are deficient. Routine use of the vitamin B complex is advocated.

*Drugs.*—Alcohol is an excellent vasodilator. Addiction may follow its use. The salicylates are excellent for the relief of pain. Habit forming drugs should not be used. The use of sulfur compounds such as sodium iodide thiosulfate,[75] or sodium thiosulfate and sodium tetrathionate[71] have been advocated. We have not seen improvement after their use.

*Oxygen.*—The inhalation or injection of oxygen does not have a rational basis in the experience of most clinics.

*Intra-arterial Injections.*—The injection of a vasodilating or antibiotic drug directly into the affected artery has been employed by some clinics for years. The advantage of placing the substance directly at the site of pathology does not counteract the danger of repeated intra-arterial punctures into a diseased artery. Priscoline has been injected in this way as have some of the other vasodilators. Marked improvement has been recorded in the occasional patient. In the acute stage in the selected patient it is of value.

*Cortisone and ACTH.*—A "sensational" recovery has been reported in the use of these substances.[31] The author has seen no improvement and many patients seem to be made worse by the use of these substances. Duryee's experiences have been similar.[16]

*Anticoagulant Drugs.*—The future control of this lesion may be possible with these substances. This problem has been discussed on pages 658 to 660.

*Specific Surgical Measures.*—Surgical sympathectomy specifically helps many of these patients. The other surgical procedures are discussed on pages 190 to 214.

C. SURGICAL MANAGEMENT.—The operative treatment of thromboangiitis obliterans is considered with the surgical management of arterio-

sclerosis and the other occlusive diseases (see pages 190 to 214). The subjects of surgical drainage and interruption of the sympathetic system are discussed on pages 191 and 199. By passing blocked arteries is detailed on pages 202 to 210. Amputation is considered in Chapter 15, page 234.

## ARTERIOSCLEROSIS AND ATHEROSCLEROSIS

**Etiology.**—It may have been our desire to limit the cause of this disease to a single factor which has delayed our recognition of the pathogenesis of these occlusive arterial lesions. At any rate, the etiology of arteriosclerosis for years has eluded investigators. Although a number of theories have been advanced, for the most part little is known about the cause of this condition. The prevailing opinion that old age, with its attending degeneration, is the cause for arteriosclerosis is no longer tenable. Delayed recognition of etiologic causes for years resulted in a defeatist attitude toward its prevention. Today, in the light of more recent evidence, it appears that other factors, such as diet and metabolism, play the major role in producing sclerotic lesions. Studies must be continued to determine the etiology of arteriosclerosis, the greatest destroyer we have to contend with today. There are two main types of arterial obliterations which in their terminal stage may appear similar but in their origin are different entirely. These two have been called arterio- and atherosclerosis.

*Arteriosclerosis* is a term used somewhat heterogenously for all types of arterial obliterations except that of thromboangiitis obliterans. The disease appears to be of a hyperplastic type beginning shortly after a person is born. It affects all the layers of the arteries to a degree.[49] It is apparently the result of intravascular tension or trauma and is dependent upon the degree of tension and therefore extends more rapidly when hypertension coexists. Even in the absence of hypertension, at times it is an irreversible process and is present to a degree in all individuals. It occurs in all types of animals who have a vascular system and in all humans despite the place or time of their living. It existed in the ancient people as it is present today and is not entirely dependent upon the diet or the mode of life. All these factors play a part, however, in the pathogenesis. It progresses more rapidly in arteries where there is stress, and therefore it will appear more often at points of friction and in vessels where more trauma exists. Its occurrence, to a certain extent, depends upon the composition of the blood. It cannot be considered always a disease until it develops an occlusion of a vital vessel. It is not synonymous, therefore, with atherosclerosis.

*Atherosclerosis*, on the other hand, is etiologically dependent upon cholesterol metabolism or dysmetabolism. Our knowledge at the present time must be considered still fragmentary, but certainly a great deal has been learned about it in the last few years. Why the lipid material is deposited upon the walls and why certain areas are predisposed to such deposits is still not clear. The reason why, in one individual, cholesterol will pass through the system and be eliminated to a large extent, while in another an even smaller amount of cholesterol will cause a deposit upon

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the wall, has not been ascertained. There are four factors which are known to be associated with the premature appearance of atherosclerosis. These are:

1. *Mechanical* —Under this heading are included such points as *increased blood pressure,* and the tendency for atheromata to form at points of *stress* in the vascular tree, such as at the origin of the intercostal and coronary arteries It is likely there is greater trauma to the intima of the vessel at such areas Countering this thought is Hueper's[30] theory that there are greater "eddies and currents" at such anatomical points of stress with slowing of the blood flow If the plasma colloids are unstable, they would tend to precipitate then at such points It appears, therefore, that the mechanical factors themselves are not primary The part that hypertension plays in the development of atherosclerosis is partially a mechanical factor, the tension being traumatic Hypertension is not entirely mechanical since it is known that in those families who have hypertension over a period of years each successive generation will have high blood pressure occur at a younger age than their family predecessor This shows that to a certain extent there is an *inherent* tendency Anatomical and mechanical variations in the origin of artery branches may be a factor The traumatic element of large or giant molecules is discussed under the metabolism paragraph

2 *Metabolism* —The greater incidence of atherosclerosis in patients who are obese, have xanthomatosis, diabetes, myxedema, and nephrosis indicates the part that metabolism, or lack of correct metabolism, plays The *serum cholesterol* varies greatly in normal subjects and depends, to a certain extent, upon the chemical method employed in its determination The technic of Bloor[5] gives a normal cholesterol value of 175 to 300 mg per 100 cc , and that of Schonheimer and Sperry[58] from 150 to 250 mg per 100 cc The ratio of the cholesterol ester to free cholesterol is consistently 3:1 It has been shown, however, that in *normal individuals* the serum cholesterol will remain the same from hour to hour, day to day, and month to month [68] In a full year's study, there was less than 10 per cent variation with the normal individuals [66,68,69] In patients who are developing cholesterosis and atherosclerosis, there will be a significantly higher and greater fluctuation in this cholesterol level than in normal individuals The part that *diet* will play in atherosclerosis has been the subject of much study For example, autopsies performed in China[66] and on the island of Okinawa,[67] where the diet is extremely low in cholesterol, have shown a similarly low incidence of atherosclerosis In one series of autopsies on Okinawa, there were only 7 per cent who had atheromata These changes were all minimal in the aorta and not a single case of coronary atherosclerosis was found A report from Finland[31] that during 1940 to 1946 there was a 50 per cent decrease in the incidence of death due to coronary atherosclerosis corresponds closely to the limitation of fats and calories in the diet of these people in those years due to the war One would have expected a higher coronary occlusion incidence during these years with the emotional stress of war acting as a precipitant That one can raise or lower the serum cholesterol by diet has been shown Thus, the American raised on cream, butter fat chocolate, ice cream, mayonnaise and other fats appears the

best dietetic candidate for cholesterosis and this statistically is true. Gutman, Watkin, and Froeb[24] dropped the cholesterol on an average of 42 mg per 100 cc as a result of a rice diet. Patients with xanthomatosis also synthesize cholesterol from other food substances more rapidly than do the normal individuals.

While we must admit that the epidemiology of atherosclerosis is in its early stage, two points can be accepted. A high level of cholesterosis leads to early and severe atherosclerosis and to coronary heart disease. The studies of Gofman *et al.*[22-23-30-31] showed that cholesterol does not exist as a simple solution but in combination with proteins and lipoids. They showed that "giant molecules," which can be determined by the ultra-centrifuge, are found in such patients. While this is disputed, the variation in the size of the molecules is suggestive and deserving of further study. Such large molecules by a continuous bombardment of the arterial walls may cause a reaction and eventually a lethal effect. Such a bombardment occurs in the normal individual 3 times a day. In those who eat excessively of fats or at too frequent intervals the deleterious effects may be compounded.

From animal experiments it is apparent that the serum lipid is the primary factor in the development of atherosclerosis. Guinea pigs, mice, rabbits, dogs, chickens and geese develop the disease when they are given a diet that has a large amount of cholesterol with fat and thiouracil added, if such feedings are continued long enough. Why such knowledge was not carried over into the parallel humans for so many years is difficult to understand. In other conditions animal experimentation is interpreted as pointing definitely to the same pattern in humans. It may be that the investigators themselves disliked to consider going on a cholesterol poor diet. It seems that the cholesterol level is not as important as the ratio of the total cholesterol to lipid phosphorus, and it is the inability of the serum phospholipid to keep pace with the rise in serum cholesterol that seems to do the damage. Davidson's[9] reports showed an increase of the molar ratio of cholesterol to the phospholipid as 1:1 in normal dogs and 5:1 in cholesterol thiouracil fed dogs in which atherosclerosis had been produced.[17] On the other side of the picture, when such animals were given intravenous detergents there was a decrease in the incidence and severity of the atherosclerosis, thus indicating that the serum phospholipids exert a stabilizing effect upon the colloidal state of cholesterol in the blood. Lecithin, which is one of the main phospholipids of the serum, is an emulsifying agent.[17]

The recent work of Gofman[22] reported in 1950 showing a high concentration of "giant molecules of cholesterol" in the blood of patients who had had a myocardial infarction was important. Using the ultra-centrifuge and a special flotation technic he separated certain lipo-protein complexes containing cholesterol from the serum. One group of complexes which he classified as Sf[22] 10-20 class was related to human atherosclerosis as evidenced by the fact that they appeared in higher concentration in the blood of patients who had had a myocardial infarction than in those who had not. On experimental animals the degree of atherosclerosis at autopsy varied directly with the height of the final concentration of Sf 10-20 molecules.

TABLE 1 —ARTERIOSCLEROSIS MENU

| Meal plan | | Amount | |
|---|---|---|---|
| | | Grams | Measure |
| **BREAKFAST** | | | |
| Fruit juice | Orange juice or tomato juice | 200 | 6⅔ ounces |
| Cereal | Shredded Wheat | 30 | 1 biscuit |
| Skimmed milk | Skimmed milk | 120 | 4 ounces |
| Butter substitute (no butter) | Vitamin-enriched margarine | 10 | 1 square |
| Bread | Whole wheat toast | 60 | 2 slices |
| Sugar (may use) | Sugar | 15 | 3 teaspoons |
| Hot beverage | Coffee, tea, Postum | — | — |
| **NOON MEAL** | | | |
| Soup | Skimmed milk pea soup | 150 | 5 ounces |
| Meat or cheese | Cold roast lamb, lean | 60 | 2 ounces |
| | Mint jelly | 25 | 1 tablespoon |
| Vegetables | String beans | 100 | ¾ cup |
| Salad | Sliced tomato | 100 | 1 medium |
| Fruit or dessert | Canned pineapple | 100 | 1 slice |
| Bread | Whole wheat bread | 30 | 1 slice |
| Butter substitute (no butter) | Vitamin-enriched margarine | 5 | ½ square |
| Hot beverage, or skimmed milk only | | | |
| **EVENING MEAL** | | | |
| Fruit cocktail | Grapefruit cocktail | 100 | ½ medium |
| Meat | Lean meats | 60 | 2 ounces |
| Potato | Baked potato | 150 | 1 medium |
| Vegetables | Asparagus | 100 | 6–8 stalks |
| | Banana squash | 100 | ½ cup |
| Salad | Fresh pear salad | 100 | 1 medium |
| Salad dressing | Boiled dressing | 20 | 1 tablespoon |
| Dessert | Lemon sherbet | 90 | ⅛ quart |
| Bread | Whole wheat bread | 30 | 1 slice |
| Milk | Skimmed milk | 180 | ⅜ pint |

IMPORTANT TO YOUR HEALTH

Avoid oysters, caviar and other roe
Serve only lean meat or fish
Use only 1 egg yolk per week, egg whites may be used as desired
Allow 1 pint or more of skimmed or buttermilk daily
Use only skimmed milk cheese such as cottage cheese Omit rich cheese, such as cream or Cheddar
Use no animal fats such as lard and suet in cooking Unless specifically restricted, olive oil, Crisco, margarine, mayonnaise, and French dressing and *other fats from vegetables and nut oils may be used* RARELY
Use vegetables and fruits as desired—prepared without butter and cream
Prepare tapioca, cornstarch, rice pudding and junket with skimmed milk and without egg yolk Whips may be made with gelatin or egg white no cream
Serve jelly, jam, marmalade, honey, molasses, syrup and sugar as desired and in place of butter
The vegetables included in this diet are asparagus, broccoli, carrots, green beans, kale, yellow squash, pumpkin, spinach, turnip greens and other greens Other vegetables—*tomato (fresh, canned or juice)*, vegetables com-

monly served raw as celery, cucumber, lettuce and cabbage, and other cooked vegetables as beets, eggplant, onions, rutabagas and cauliflower

Take Vitamin A as ordered

*May Eat*

*Soups* Bouillon, fat-free vegetable soups, vegetable broth and soups made with skimmed milk

*Meat, Fish and Poultry* Lean meats broiled, roasted, baked or boiled

*Eggs* Egg whites as desired, not more than 1 whole egg weekly

*Milk and Milk Products* One pint or more of skimmed milk or buttermilk, cheese made from skimmed milk

*Vegetables* All cooked or raw, especially the green and yellow vegetables rich in Vitamin A, namely beet greens, chard, spinach, carrots, kale and mustard greens

*Fruits* All fruits raw, cooked, dried and canned. Use citrus or tomatoes daily

*Salads* Any raw or cooked fruit or vegetable salad and gelatin salads. Serve with boiled or low fat dressings such as those containing mineral oil (refined), lemon juice, spices, vinegar, ketchup, etc

*Cereals* All cooked or dry cereal, macaroni, spaghetti and rice, serve with skimmed milk

*Breads* Whole wheat, enriched white, rye bread or rolls, graham and soda crackers

*Deserts* Fruits, tapioca, cornstarch, rice, sago, junket puddings made with skimmed milk and without egg yolk, fruit whips made with egg whites, gelatin desserts, angel food cake, macaroons and egg kisses, water ices

*Concentrated Sweets* Jam, jellies, marmalade, honey, molasses, maple syrup (instead of butter) and sugar as desired, hard candies

*Beverages* Tea, coffee or coffee substitutes, tomato juice, fruit or vegetable juices

*Do Not Eat*

*Soups* Cream soups

*Meats* All glandular organs as liver, brain, kidney, sweet bread, pork and very fat meat, fat fish, fish roe

*Milk and Milk Products* Whole milk, cream, Cheddar, Swiss and all rich cheese and cheese spreads, very much butter and butter substitutes

*Eggs* Egg yolks

*Breads* Hot bread, pancakes, waffles, coffee cakes, muffins, doughnuts

*Deserts* Any made with cream and egg yolks, pies, frozen creams, rich cakes and cookies

*Concentrated Fats* The excessive use of fats in any form as salad dressings, olive or vegetable oils, suet, chicken or pork fat

*Miscellaneous* Rich gravies, olives, nuts and avocados

The further relationship between the cholesterol lipid phosphorus ratio of serum and the concentration of these molecules has not been sufficiently investigated. It has been shown that adherence to a diet restricting cholesterol to 200 mg and fat to 50 gm or less daily for periods of four weeks consistently lowers the concentration of these giant molecules. While it has been shown[37] that a diet of 50 to 800 mg per day shows no correlation with the serum cholesterol level, when the cholesterol and the fat are eliminated entirely from the diet a rapid decline in serum cholesterol level occurs. Fat appears to be as important as cholesterol to maintain a high serum cholesterol. Thus the work which proved that diet did not influence the cholesterol level was based on *insufficient restriction* of both fat and cholesterol. It follows therefore that in those who

are atherosclerotic there is a definitely diffierent level in which these basic fat and cholesterol levels in the serum must be maintained if deposit on the intima of the arteries is to be prevented. In the near future, one may be able to select in early life those who will develop atherosclerosis merely by a careful study of the "giant molecules" or the phospholipid-cholesterol serum levels, and to institute in them a therapeutic measure beginning at the time they are born which may retard or entirely negate the degenerative changes to which they already are inherent.

*Further Thoughts on Cholesterol Mechanism* —Why does cholesterol fall from its colloidal solution at intermittent intervals, and how is the colloidal suspension maintained in the first place? Another question is, why does this suspension apparently hold itself in check for about one-third of a person's life and then fall into dysfunction? Why is it also that at some anatomical point in the circulatory system, usually where the direction of the arterial flow changes, the suspensions are deposited more often, while one might think that at such points with the quick changing of the direction of the flow that the colloidal suspension would be carried past?

*Cholesterol Metabolism* —We have both exogenous and endogenous cholesterol in the body and these are derived from the intestines with the fatty acids Part of this free cholesterol is excreted in the feces as coprosterol Some of it becomes esterified and is absorbed and carried to the liver The cholesterol then is "freed" and the fatty acids enter into combination with phosphoric acid and choline to form lecithin Some of the fatty acids become acetates and aid, probably, in cholesterol production The cholesterols become esters in the liver Choline, too, comes from the intestine Cholesterol then joins with the protein in the parenchyma of the liver as a complex molecule As a result of all this chemical change there are released into the blood stream four types of cholesterol

(*a*) The endogenous, which is the synthesized cholesterol

(*b*) The exogenous is the cholesterol that is ingested and not made into an ester

(*c*) The giant molecule, cholesterol, and protein molecule (as described by Gofman) [22]

(*d*) The cholesterol ester, which comes from the liver and the intestines

Some of this cholesterol is secreted in the bile Thus, we have an enterohepatic circulation The cholesterol is removed from the intestines, goes into the liver, the blood stream and then back to the intestines as bile For some reason, perhaps due to the change in the permeability of the tissues to the colloidal balance, some abnormal pressure, or an abnormal concentration, some of this substance is deposited in and on the intima of the arteries There appears to be a critical level in the blood stream which the cholesterol must reach before it becomes a pathologic entity If the cholesterol concentration is kept below that entity, then one may be able to prevent the deposit of cholesterol on the intima This is true particularly in those in whom a cholesterol deposit may be suspected or expected Perhaps the level of such a patient should be even a lower one If 250 to 800 mg per day of cholesterol is ingested there will be no change in serum cholesterol If, however, a fat-free, cholesterol-free diet is instituted the serum cholesterol level reduces rapidly It seems proven,

also that fat is as important as cholesterol in lowering the serum cholesterol value[22] Perhaps some natural lipotrophic substance similar to insulin in sugar metabolism will free us of this killer cholesterosis Maybe we have this substance already i e heparin[23]

*Effect of Nicotine* —One fact that has not been considered of sufficient importance is the possible effect of nicotine on the deposit of atherosclerosis In the non-smoker the incidence of sclerosis is definitely lower This may be the factor which either disturbs the colloidal balance of cholesterol or increases the permeability of the blood vessel permitting the deposit to appear One other thought is that the sugar and the glucose element by means of which the fat must burn is inactivated in some way possibly again due to nicotine We do know that in diabetics the addition of nicotine smoking releases glucose from glycogen and changes the sugar metabolism greatly Perhaps a diabetic-like state is set up temporarily with the smoking and the failure of sugar fat metabolism results in the failure of cholesterol metabolism That the normal lipid metabolism can vary in all and does vary in many with a resultant atherogenesis appears proven[22] A shift to a normal lipoprotein pattern can be caused by the injection of heparin The potential effects of this fact diagnostically and therapeutically appear unlimited Our present concepts are primary

From these factors metabolism looms large in the development of atherosclerosis and perhaps is the only factor It has been significantly impressive for our Clinic to put all patients with arterial or cardiac lesions on a diet extremely low in cholesterol and fat as a therapeutic measure (See Table 1 )

(3) *Endocrine Factor* —The sex difference in the development of atherosclerosis is well pointed out in Table 5 on page 184 The lower incidence in the female in both coronary and end artery changes is well known to all and is shown in Tables 2 to 6 A somewhat incongruous finding however is the fact that obesity is more common in the female and yet the male more often has the atheromatous changes It is pointed out also that in similar patients with the same background the same diet and the same age similar pathologic changes necessarily do not occur While brothers may not have the same lesion the reverse more often is true Whether sclerotic changes occur because of heredity anatomical factors, aberration of the endocrine and metabolic function or through diet is not too well established but that there is an endocrine background factor seems quite definite The apparent good results of the use of endocrine glands therapeutically also points to this being important

(4) *Heredity* —Heredity has been discussed before but without question a susceptibility to the disease can be handed down from one generation to the next Filert s[17] report in which in one family the father had a coronary occlusion in his sixties his son a coronary at forty five and the grandson one at twenty-eight is not too unusual and other physicians have reported similar examples of an endocrine or congenital basic antithesis to the disease Occlusion can be expected to occur at an earlier age and to be more extensive in the children of males who have advanced sclerotic changes The same precept follows for the chronic complications which follow these changes.

From this discussion it is apparent that a great deal is not known about the etiology of atherosclerosis, but since this subject was first written five years ago, great progress has been made[52]

TABLE 2.—INCIDENCE OF ARTERIOSCLEROSIS OF THE LEG ARTERIES IN WORKERS OVER FORTY YEARS OF AGE[39]

| Age groups | Total in group | *Disease revealed by roentgen examination* — *With arteriosclerosis* — Number | Per cent | Total with calcification | Number with calcification only | Disease revealed by other evidence |
|---|---|---|---|---|---|---|
| *Men* | | | | | | |
| All Groups | 305 | 141 | 46 | 135 | 115 | 6 |
| Workers who stand | 89 | 38 | 43 | 35 | 30 | 3 |
| Workers who walk | 129 | 64 | 50 | 62 | 54 | 2 |
| Workers who sit | 40 | 16 | 40 | 16 | 15 | — |
| Workers who climb stairs | 47 | 23 | 39 | 22 | 16 | 1 |
| *Women* | | | | | | |
| All Groups | 231 | 46 | 20 | 42 | 32 | 4 |
| Workers who stand | 107 | 22 | 21 | 20 | 16 | 2 |
| Workers who walk | 56 | 10 | 18 | 9 | 6 | 1 |
| Workers who sit | 66 | 13 | 19 | 12 | 10 | 1 |
| Workers who climb stairs | 2 | 1 | — | 1 | — | — |

TABLE 3.—SEX AND AGE INCIDENCE OF ARTERIAL IMPAIRMENT[39]

| | *40–49 years* | *50–59 years* | *60 Years and over* | *Total* |
|---|---|---|---|---|
| Men | 27% | 55% | 77% | 46% |
| Women | 8% | 26% | 56% | 20% |

TABLE 4.—INCIDENCE OF ARTERIAL INVOLVEMENT AMONG MEN AGED FORTY TO FORTY-NINE[39]

| | *Total* | *With arterial involvement* — *Number* | *Per cent* |
|---|---|---|---|
| Workers who climb stairs | 29 | 11 | 38 |
| Others | 108 | 23 | 21 |

TABLE 5.—INCIDENCE OF CALCIFICATION—BOTH SEXES[39]

| | *Total* | *With calcification* — *Number* | *Per cent* | *With 3 and 4+ calcification* — *Number* | *Per cent* |
|---|---|---|---|---|---|
| Men | 305 | 135 | 44 | 41 | 30 |
| Women | 231 | 42 | 18 | 3 | 12 |

TABLE 6.—INCIDENCE OF PLAQUES BY AGE GROUPS[19]

| Age group (both sexes) | Number with calcification | Per cent with plaques |
|---|---|---|
| 40 to 50 years | 10 | - |
| 50 to 60 years | 1 | 15 |
| 60 years and over | 63 | 29 |

*Sex and Age.—Research in Arteriosclerosis.*—Two out of every three of us will die of cardiovascular disease or one of its complications. The small amount of money being expended for research in this field, considering the mortality rate in comparison to that spent on poliomyelitis, carcinoma and other allied conditions, is alarming.

TABLE 7.—MORTALITY RATE AND RESEARCH EXPENDITURE

| Diseases | Deaths | Amount spent per death |
|---|---|---|
| Poliomyelitis | 2 0 1 | $1,200 00 |
| Tuberculosis | 80 000 | 172 00 |
| Cancer | 217 000 | 66 22 |
| Cardiovascular diseases | 763 000 | 9 62 |

Figures supplied by the New York Heart Association[62]

None of us are quite certain whether arteriosclerosis can be prevented, as atheromatous degeneration and cholesterol dysmetabolism are associated with degeneration of nearly any part of the body. This disease probably was planned by Nature to prevent over population of the world, but the study of this problem still is indicated. This disease combines with war and epidemic to maintain the ratio between birth and death.[5]

In an analysis of 536 persons of both sexes over forty years of age, we (Lake, Pratt, Wright)[19] found arteriosclerotic changes in 46 per cent of the males in contrast to only 20 per cent of the females. When the degree of sclerotic changes, as evidenced by the occurrence of plaques is considered, then the tendency of males to develop the condition becomes even more apparent. While 30 per cent of the males had 3 and 4 plus calcification, only 12 per cent of the females fell into this classification. Our study confirmed the fact that the incidence and severity of arteriosclerosis progress with increasing age. We found an increasingly higher percentage of individuals with arteriosclerotic lesions in the older age groups.

These findings are summarized in Tables 2, 3, 4, 5 and 6. In our study we found it difficult to correlate the sex and occupational differences with age and degeneration. We are certain that the lesions begin in early life. One of our patients had calcification in a leg vessel after trauma at the age of four (Fig. 35).

*Liver Changes.*—Since the liver plays a part in lipid and lipoprotein metabolism, diseases or severe changes in the liver can play an important

part in the development of atherosclerosis This problem has been discussed under the metabolic considerations on the previous pages The possibility of lipotrophic agents being activated by the liver has therapeutic implications

*Focal Infections*—Focal infections play a part in initiating signs, if not in the actual onset of the condition, at least in continuing the process once it has been initiated These foci may precipitate symptoms Such focal infections are usually found in the mouth, throat or sinus sites, but dental caries is the most frequent offender The patient with roentgen evidence of defective teeth will usually continue to have severe symptoms of arterial failure until the foci of infection are removed

*Occupation* —There has been no correlation between the patient's disease and his work An acute or continuing trauma to a patient who is developing arterial changes may precipitate an occlusion incident Over-use of a part of the body may call for more blood than the part can supply Claudication or arterial failure may occur. Our efforts to show greater incidences of arterial occlusion in patients doing strenuous work did not succeed See Tables 2 to 6

*Nicotine* —Nicotine is a factor both in the cause and acceleration of symptoms of occlusive arterial diseases (see pages 160, 165). It is reiterated that total abstinence from tobacco should be prescribed for every individual who is over forty years of age and has a familial history of any cardiovascular disease or diabetes or shows any evidence of having the condition

*Diabetes* —The importance of diabetes as a factor in arteriosclerosis has been mentioned (see page 216) In those with glucose imbalance and faulty fat metabolism, arteriosclerosis will be apparent after a minimum of four to five years.

*Initiating Causes* —An injury may initiate the occlusion Any skin break, infection, pressure point, ingrown toenail, or burn may activate the symptoms of circulation failure The exposure to cold or sunburn, as well as poor hygiene, may contribute to the underlying cause

**Symptoms** —The symptoms of arteriosclerosis are similar to those of all occlusive arterial diseases of the obliterative type, as outlined under thromboangiitis obliterans (see pages 160 to 161)

The arteriosclerotic patient usually but not necessarily is older than the patient with thromboangiitis obliterans The lesion may be very slow in making its appearance, claudication and the rubor-pallor (RP) sign may be delayed for a long time The first symptom may be the failure of some simple infection, such as an ingrown toenail, to heal In some cases, a sudden closure of a previous partially occluded vessel may cause an acute arterial occlusion, with symptoms similar to those following an embolus

The symptoms and finding of arteriosclerosis elsewhere in the body are suggestive

Arteriosclerosis should be expected in patients with diabetes mellitus in spite of age

Claudication develops with increased occlusion and may progress to the point where the individual must stop walking completely

*Trophic Changes.*—The foot may atrophy markedly with trophic changes and become shiny and white. There will be a loss of hair. In some cases there is an accompanying edema, which may be due to the patient's keeping the foot dependent. This edema, by its local pressure, produces a mild anesthetic effect and gives some patients slight relief from pain.

*Infection.*—With the failing blood supply, the part becomes susceptible to infection. An unexpected or continued infection is suggestive evidence of arterial failure. When inflamed or infected, the leg becomes red and then dusky. If the infection is not controlled, necrosis results early with areas of bleb formation, purulent collections and ultimately gangrene which begins in the skin. When drainage is adequate, the redness may disappear. The subcutaneous tissues and tendons become involved early, either with infection or by avascularity, and secondary osteomyelitis develops later. With loss of blood supply, rarefying osteitis is usual.

**Diagnosis.**—Arteriosclerosis or atherosclerosis may involve any or all parts of the body. The factors essential for making a diagnosis include:

1. Arterial insufficiency due to arteriosclerosis or atheromata in one or more of the extremities.

*Symptoms of arterial insufficiency are*

(a) intermittent claudication
(b) pain at rest
(c) pallor on elevation
(d) absence of pulsation
(e) rubor on dependency
(f) coldness
(g) trophic changes
(h) failure of a simple skin break or infection to heal, and
(i) gangrene

These changes must be present in the absence of trauma, embolism, arterial thrombosis, or congenital anomalies.

2. Other factors favoring the diagnosis are

(a) The individual smokes.
(b) The patient is nearly forty years of age.
(c) Evidence of arteriosclerosis or atheromata elsewhere in the body.
(d) Calcification of any part of the vascular system as shown by roentgen ray or clinical examination.
(e) Diabetes mellitus.

*Chemical Tests for Detection of and Tendency Toward Atherosclerosis.*—While incomplete both as to accuracy and interpretation, there are three methods of determining atherosclerosis.

1. The optical ultracentrifuge of Gofman.[22,23] This method spins the blood at 52,000 revolutions per minute and thus separates the pathologic lipid and lipoprotein molecules. It is costly and not generally available at present.

2. Blood serum phospholipid-cholesterol ratio of Kellner. Patients with atherosclerosis have a subnormal ratio of blood phospholipid to cholesterol.[24]

3. Cholesterol partitioning procedure. This measure splits the cholesterol protein molecular complex in blood serum by saponin. An estimation of the unstable versus the protein bound cholesterol thus is made.[44]

According to Morrison, the latter two methods are accurate in 3 out of 4 patients [48]

These laboratory tests are not available generally in standard laboratories at this time Better and more accurate tests will be devised

## GENERAL AND SURGICAL TREATMENT OF OCCLUSIVE ARTERIAL DISEASES

### (Thromboangiitis Obliterans and Arteriosclerosis or Atherosclerosis Obliterans)

The general principles of the therapy of occlusive arterial diseases were discussed in detail under Thromboangiitis Obliterans (see pages 162 to 177) The same methods apply also in arteriosclerosis obliterans A summary in outline form is given below

I **Prophylactic Measures.**—1 Discontinue all *smoking* Spasm of the major affected artery and the collateral vessels always accompanies arteriosclerosis Nicotine causes a release of a pituitary gland hormone into the blood stream The smoking of one or two cigarettes releases this substance in sufficient quantities to produce constriction of the coronary arteries in the dog and likely in man [63] The spasm of small vessels caused by nicotine makes smoking and occlusive arterial disease incompatible That is why it is imperative that smoking be stopped at once and permanently in all those with a predilection to occlusive arterial disease, as determined by the familial history, and in all those in whom symptoms of the disease become apparent

2 Elimination of the use of *vasoconstricting drugs* like ergot or adrenalin

3 Special *foot care* to prevent skin breaks, pressure points, burns, ingrown toenails, bunions, corns, or fungus infections

4 *Stimulation* of the remaining *collateral vessels*, relaxation of all spasm, and efforts to develop new vessels

5 *Anticoagulants* —In all of those who are expected to develop obliteration, the use of anticoagulant therapy should be considered The future use of these substances may be revolutionary in the therapy of these lesions The fact that heparin and heparin-like substances affect cholesterols gives further strength to this point The main function, however, will be the prevention of clotting of the blood stream where it is slowed in partly occluded vessels

6 *Use of the part* below the claudication stage helps stimulate collateral circulation In this respect, moderate walking or swimming is to be recommended With this activity there should be adequate periods of rest

7 *Baths and Heat* —The use of warm *soaks* and *Sitz baths* are important This is one of the reasons for the reported efficacy of the various Spas in Europe through the years These resorts had as their real value, rest, warmth, and the general feeling of well-being which follows baths, relaxation and any physical change in one's environment

8 *Drugs* The use of drugs has been disappointing in general, but may be of value in some patients The action of some of the sympatholytic and

adrenolytic drugs will be discussed in the chapter on page 487. Aspirin and whiskey seem to be the best of the drugs for relief of pain.

9. *Diet.*—In patients who have arteriosclerosis and/or artherosclerosis a low fat, low cholesterol fat intake is indicated. We routinely reduce the fat intake to 20 grams daily (see pages 180 to 181). A sample diet which we advocate for all of our patients in this category contains this

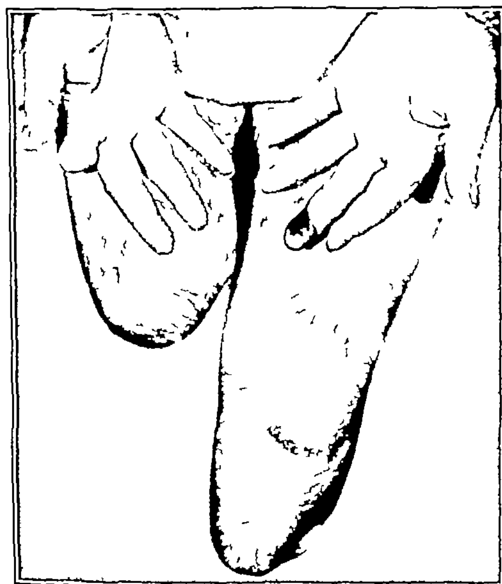


Fig. 30.—Patient with thromboangiitis obliterans. Patient continued smoking. Loss of both limbs, beginning loss of digits of the hand. Complete economic liability at age of 40.

low cholesterol and fat intake. It is not difficult to accustom one to such a diet. It is wise to add a vitamin A component to the diet to replace necessary vitamin A which the patient normally would obtain from a higher fat intake diet.

10. *Lipotrophic Substances.*—While no one is certain of the lipotrophic potential of some of the choline group, it is felt that it is wise to use these

substances in combination with the low cholesterol fat diet A combination of choline and inositol, using 3 grams of choline and 1½ grams of inositol a day, may be given It is thought that these substances may be either protective or therapeutic and possibly both Choline chloride in doses of 4 grams daily has been reported to be toxic in certain individuals Nine per cent of patients reported by Gates, all of whom were diabetics, developed some toxicity to the drug, the toxic manifestations being edema of the lower extremities in 5 of the 9 treated patients

11 *Endocrine Replacement and Stimulation Therapy* —A basal metabolism test should be run on all these patients If it is low, the use of a small amount of thyroid routinely is indicated This therapy should be controlled by having the patient take his pulse at bed rest in the morning If there is an abnormal rise, the thyroid should be decreased or stopped In addition, use of the estrogen and androgen hormone extracts is indicated for those deficient The hormones should be discontinued ten out of every thirty days In the female, this cessation should be prior to the menstrual period The validity of this endocrine factor remains uncertain It is based partly on the sex variation in the incidence of arterial disease (see pages 183–184)

II **Active Treatment.**—*Active Treatment* is threefold consisting of (1) general measures aimed at improving the general condition of the patient, (2) local measures directed toward alleviating the local lesion, and (3) attempts to revascularize the limb

A General Measures Include

1 Adequate antibiotic therapy to eradicate secondary invading organisms whose presence is determined by bacteriologic studies The correct antibiotic to which the organism will respond should be determined by sensitivity tests In the modern hospital laboratory, this report can be obtained in a few hours

2 Correction of constitutional defects in the cardiac, renal, or pulmonary systems and deficiencies in blood, vitamins, or proteins

3 Anticoagulant therapy

4 Small repeated blood transfusions may supply the blood factor needed to overcome an infection that cannot be controlled by other methods

B Local Treatment

This includes care of the infection or skin break to keep it localized, draining, and to prevent it from undermining This is accomplished by the following

1 *Soaks* —Saline soaks daily *for one hour are important* These soften any collection and take the place of a wet dressing More recently we have used the whirlpool soak which debrides in a gentle manner by the agitation of the solution To this solution is added a sulfonated detergent, pHisoHex, which has Hexachlorophene in it This combination works effectively

2 *Debridement* —The soak or whirlpool is followed by careful removal of any loose sloughs or drainage material This must be done without pain or blood-letting, for occurrence of either pain or blood indicates that viable, and not *dead*, tissue is being disturbed

*In the use of enzymes for debridement* streptokinase and streptodornase have been found to have fibrinolytic properties Trypsin has similar action The application of these substances to necrotic areas may aid in the separation of the live from the devitalized tissue These physiologic curettage substances have not acted clinically as well as expected Their potentialities exist for the future and it may be that an improvement or change in their type or strength may be the answer to the need for a substance to separate nonviable from viable tissue.

3 *Antibiotic Therapy* —This may be applied locally to destroy the causative organism The application of azochloramide (N-N-Dichloroazodicarbonamidine in Triacetin' 1 500) may help at this stage

4 *A mild fungicide soak* daily will help control the usual fungus contaminants. A 1 15 000 to 1 25 000 solution of potassium permanganate for twenty minutes is recommended

5 *Incision and drainage* of any localized collection of pus

6 *Bloodless removal* of any dead toe or tendon when it is fully demarcated

7 *Removal of any undermined skin* will eliminate pocket formation

C RECIRCULARIZATION OF THE EXTREMITIES —This includes sympathetic blocks sympathectomy and by passing or removing blocks in the arteries. This is discussed in subsequent pages See page 198

**Complications** —In addition to the general local and revascularization measures outlined above prompt local surgical treatment is required to stop as soon as possible the complications to which patients with occlusive arterial lesions are subject

Included among the complications are (1) simple infections (2) cellulitis (3) tenosynovitis (4) osteomyeltis and (5) gangrene

Each of these if not properly controlled promptly may lead to a major amputation

1 *Simple Infections* —Patients with obliterative diseases cannot withstand even a *mild infection* A day s delay in the evacuation of a small collection of pus beneath or near the nail may cause the development of a gangrenous toe the next day and require a major amputation on the third day This pus accumulation exerts pressure against the tiny remaining collateral vessels that are still functioning and obstructs them Thus the treatment of local infections in the arteriosclerotic patient requires an earlier but less radical surgical drainage than would have been performed had the patient not had the underlying vascular lesion

2 *Cellulitis* —The same early drainage principle applies to cellulitis which usually arises from a toe and spreads over the dorsum and more rarely on the plantar surface of the foot Trophic or perforating types of ulcer occur with this cellulitis The infection spreads slowly to the ankle Although cellulitis over the foot may be controlled for weeks or months once it reaches the ankle it may extend up the leg in a single day In such cases early drainage is necessary

When such a patient is seen for the first time and the infection is spreading, adequate chemotherapy with *selection* of the correct antibiotic to which the organism is susceptible and local soaks should be initiated for twenty-four to forty-eight hours If the infection is localized the drainage



FIG 37 —Gangrene following mild trauma in a patient with arteriosclerosis



FIG. 38 —Mild trauma in a patient with occlusive arterial disease resulting in gangrene

*A*

*B*



*C*

FIG 39—4 Gangrene with extending infection. Treatment included drainage antibiotics sympathectomy and self amputation. *B* Necrosis of the great toe with self-amputation The bone has been rongeured away *C* End result is a serviceable limb. (Pratt courtesy of Am J Surg)

should not be delayed If the status of infection remains the same without advance or if there is some subsidence of the process, this conservative regimen of chemotherapy and drainage should be continued If the condition progresses despite such a regimen, amputation must be considered While the surgeon is making his decision, it is imperative that the patient remain off his feet and be treated with all of the conservative measures previously outlined, including the anticoagulants The anticoagulant effect may be counteracted before amputation.

3 *Tenosynovitis* —This is a frequent complication of vascular infection. It is usually of the avascular necrotizing type, resulting in gangrenous tendons and sheaths Treatment includes incision and drainage with an opening into the tendon sheath If the tendons are necrotic, they should *not* be excised until their demarcation is complete. Such tendons left long will serve as drains If cut away, they may retract up the sheath and lead to the development of a necrotic focus elsewhere When tenosynovitis is present, the limb should always be examined by roentgen ray for bone involvement

4 *Osteomyelitis* —Osteomyelitis complicating arteriosclerosis, like the osteomyelitis from other causes, may require an ample incision if there is an active infection, with saucerizing of the bone involved When the patient is first seen, the therapeutic program may be conservative for twenty-four to forty-eight hours Rarefying osteitis should be differentiated from osteomyelitis (See Figure 40 )

Conservative therapy will be effective in most patients without advancing bone infection The roentgen ray appearance may be deceiving for it may show marked and multiple destructive bone lesions Many patients are able to walk and remain conservatively active on such limbs for years Such patients, from time to time, may develop local infections that require chemotherapy or local drainage If the physician watches them carefully and treats them conservatively, they may go on indefinitely or for many years If a large incision is made in treating these lesions, necrosis usually results with later gangrene

These patients may have to be treated with penicillin or other chemotherapy every few months Conservative therapy keeps patients walking on their feet longer than radical treatment.

*To the patient*, no prosthesis—no matter how perfect—can replace any part of his own foot on which he can stand or walk

The patients with advanced occlusion should be considered like the tubercular or the advanced cardiac patient We do not expect to cure their lesions necessarily, as the disease is a degenerating and progressive one Our goal is to arrest or stay the progress of the disease When we keep these patients walking on their own or part of their own feet, we keep them both happier and living longer

Care must be taken to prevent undermining of the soft tissues around a granulating center With reduced blood supply and disuse, the bones in the foot lose their calcium content and develop rarefying osteitis and osteoporosis At times, the roentgen ray appearance of the bone with osteitis or osteoporosis resembles the bone destruction of infection In osteitis associated with avascularity, the bone, while decalcified, retains its perios-

teal contour and the medullary lines although reduced are present In osteomyelitis, there is actual bone destruction with frequent soft tissue swelling or accumulations Old or recent sequestra may show The roentgen ray picture is only a helpful diagnostic factor and should not be permitted to dictate the course of treatment to be followed



FIG. 40.—X ray shows rarefying osteitis with some areas of osteomyelitis. From the x-ray appearance of such a foot one would expect that the part would be lost This patient has been active on his feet for fifteen years despite extensive bone destruction (Pratt courtesy of Am. J Surg )

Such conservative therapy is not intended for infections which are not controllable nor for infections caused by gas gangrene bacilli. In the latter cases amputation may be lifesaving and cannot be postponed The differentiation between gas gangrene infection and the mere presence of the gas organism is important These organisms can be cultured often

where there is no infection The symptoms caused by the infection of these organisms, however, are severe and serious and have been discussed on pages 238 and 457

**Sanitarium Care** —It took centuries before physicians realized that the proper care of the tubercular, the poliomyelitic, or mentally ill patient required institution or sanitarium care extending not over a period of weeks or months, but for years, if improvement or arrest of the disease was to be expected Before such institutions were provided, many of the mentally deficient and ill patients were confined in prisons It is not long since Edward Livingston Trudeau started the first tuberculosis sanitarium in 1884

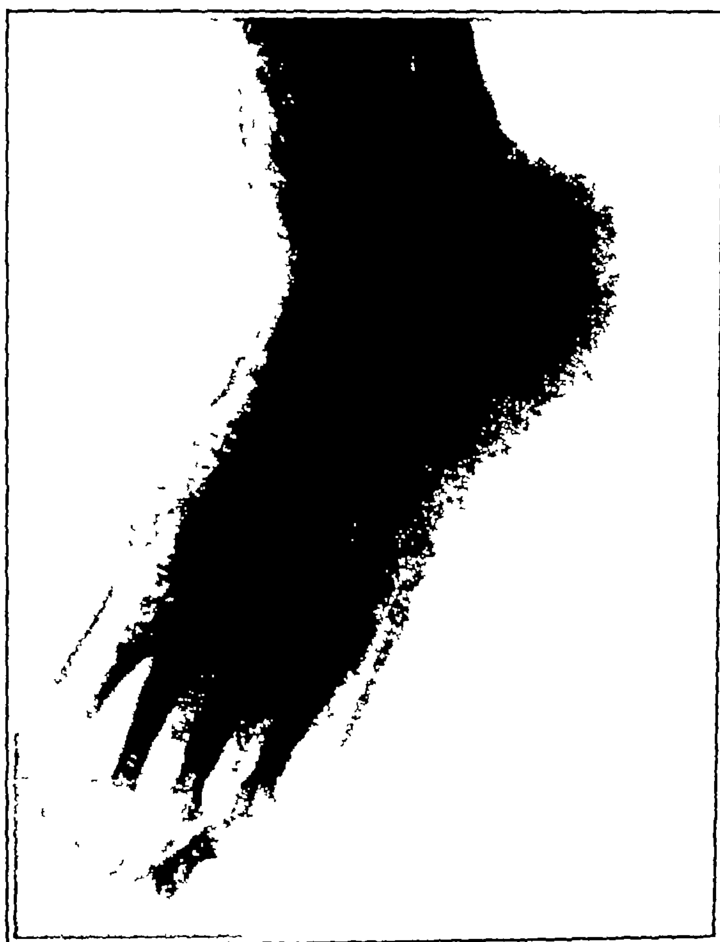


FIG 41 —X-ray showing osteomyelitis and osteitis Patient has lost part of his foot but is still walking after eleven years

The great strides made in the management of tuberculosis are a direct outgrowth of the recognition of the need for sanitarium care in the treatment of the disease We are, today, in the same stage in the management of cardiovascular diseases The patient with a mitral stenosis can have his disability so reduced with medical and surgical care over a long period of time that he can live with his disease for an indefinite period The patient with a congenital arteriovenous aneurysm may need multiple operations and a lengthy hospitalization to restore him to health, followed by his rehabilitation to some type of work which he is capable of performing.

The same is true in diabetes mellitus Not only is it necessary that the care be given in order to actively treat the condition but at the same time the patient must be taught his limitations learn to know his disease and in the words of Joslin to the diabetic to live with it

We will go a long way in the control of the patient with a cardiovascular disease and his disability only when it is recognized that an extended period of care is necessary and institutions are provided where this care can be obtained At present when an arteriosclerotic patient with a gangrenous toe is in a ward of a general hospital for a long time the surgeon is under pressure from the hospital authorities and the house staff to complete the process one way or another in order to get the patient out of the hospital

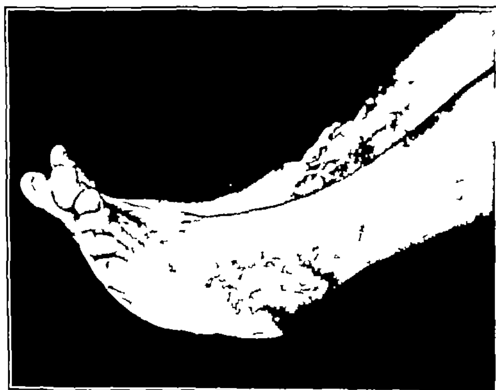


Fig 12 – Trophic changes and ulcerations in an arteriosclerotic extremity Arteriosclerotic lesions are not always in terminal vessels

In many instances this precipitates a decision to perform a major amputation. Such a situation should not be allowed to exist It is certain that many patients have lost their limbs due to just such practices

A decision for amputation should never be made on an economic or time basis *Hospital superintendents are not free entirely of responsibility* for some of the applied pressure It is understandable of course that in a general hospital one cannot keep medical or surgical beds for an indefinite period of time We know for certain however that many limbs can be saved by continuing conservative measures in treating a local toe lesion if the treatment is carried on for a sufficient length of time

It is our considered opinion that such institutions can render better care to more individuals if they are privately and not *governmentally*

*managed* It is recognized that financial help but not direction from the government may be necessary.

**Dressings** —Many wounds do well exposed to the air If redressing of an infection or necrotic area on the extremity of a patient with obliterative vascular disease must be performed, a rigid aseptic technic is necessary The diminished blood supply reduces the resistance of the tissues

The invasion of the wound by a virulent contaminant may turn a satisfactorily progressing lesion into a rapidly spreading gangrene Streptococci can be cultured from most of these sclerotic ulcers

Wet dressings commonly used in infections cannot be applied to the infections of patients with obliterative vascular diseases The constant wet dressing inevitably cools, and a vasoconstriction or spasm results in the remaining small collateral vessels The wet dressing also causes a local maceration

A warm foot soak for one hour twice a day in mild salt solution combines the advantages of the wet dressing, the softening, localizing, and removal of the exudate, without cooling or maceration The whirlpool soak has been discussed.

**Drugs** —Other drugs which have been tried by mouth are of questionable value Lugol's solution, papaverine hydrochloride ½ grain (0 032 Gm ), the iodides, and "Prostigmine" are examples of such drugs

In addition to the general and local measures so far discussed, a third part of the therapy is important, namely, attempts at recircularization

C. Recircularization of the Extremities.—This includes:

(*a*) *Simple Medical Measures* —Simple measures, such as walking and swimming below the claudication stage, stimulate collateral circulation Warm baths and posture changes also help The other mechanical gadgets, such as suction pressure or venous occlusion machines, have not contributed much in the opinion of many clinics, and in our Clinic, they have been discarded. There is no get-well-the-easy-way or with a push-button in this disease

(*b*) *Medicine* —Drugs have been described as stimulators of the collateral circulation, but no ideal one has been developed The amount of expensive literature that arrives daily on a doctor's desk telling of vasodilating drugs must make all wonder in what direction our research is headed Intravenous ether and novocain, the sympatholytic and adrenolytic drugs, dibenamine, tetraethylammonium chloride and Priscoline can block the sympathetic ganglia. Unfortunately, any such generally acting drug must act equally on every ganglion in the body The available blood supply in the body is a definite and settled amount, and when extra blood is supplied to one limb, it must be drawn from other areas.[11] Thus a surgical sympathectomy on one leg causes slight cooling of the other three extremities A generally acting drug draws blood from all areas to supply every other place If enough of the drug is taken to paralyze the sympathetics, then hypotension follows and the tendency to thrombosis thus is increased The action of these drugs on the sympathetic system is not questioned, but from the occlusive arterial standpoint, reliance alone on them may be harmful Temporarily, and for diagnosis, they are of value

(c) *Surgical Sympathectomy*.—The sympathetic system is still a poorly understood one. It functions as a defense mechanism in which a reflex vasoconstriction occurs in response to the stimulus of any trauma. This trauma may be a gunshot, a stab wound, or a bruise, in which case Nature attempts to cause that vessel and the collateral vessels to go into spasm both to stop hemorrhage and to squeeze out blood from the artery to prevent clotting. Thus, if the continuity of the circulation can be re-established, these vessels are capable again of transporting blood. The trauma also may be an intra-arterial plaque, clot or calcium deposit on the intima, originating the afferent stimuli to the ganglia which in turn send out efferent stimuli to throw that vessel and others into spasm. Whether one accepts a neurogenic reflex theory or believes a circulating spastic substance causes this reflex mechanism makes little difference because we know it occurs. The breaking up of this reflex syndrome materially aids the patient. Freeman[11] and others have said that sympathectomy in certain cases breaks down the glomus arteriovenous shunts and may

TABLE 7a.—SYMPATHECTOMY FOR OCCLUSIVE ARTERIAL DISEASE

| | |
|---|---|
| Males | 65 per cent |
| Females | 35 per cent |
| Average age | 52 |
| Sympathectomy—left side | 33 per cent |
| Sympathectomy—right side | 28 per cent |
| Sympathectomy—bilateral | 39 per cent |
| 100 patients | |

TABLE 8.—SYMPATHECTOMY FOR OCCLUSIVE ARTERIAL DISEASE

*Mortality Sympathectomy*

| | | | |
|---|---|---|---|
| 211 | 2 Deaths | | 0.4 per cent |
| Cause of death | Liver failure | 1 | |
| | Leriche's syndrome | 1 | |

TABLE 9.—SYMPATHECTOMY AND AMPUTATION INCIDENCE

| | |
|---|---|
| Total sympathectomies | 211 |
| Total analyzed | 100 |
| No amputation | 87 per cent |
| Amputation | 13 per cent |
| Amputation performed with sympathectomy | 2 per cent |
| No ganglia pathologically | 2 per cent |
| Amputation incidence after sympathectomy | 9 per cent (corrected) |

TABLE 10.—RESULTS OF SYMPATHECTOMY IN OCCLUSIVE ARTERIAL DISEASES (100 patients)

| | |
|---|---|
| *Unimproved* | 20 per cent |
| Amputations | 13 per cent |
| Continued claudication | 7 per cent |
| *Improved* | 80 per cent |
| Ulcers healed or claudication reduced | |

precipitate gangrene In our experience this gangrene has never occurred and this experience is based on over 200 sympathectomies for arterial occlusion In all of our patients, we have not been able to prevent gangrene, but we believe this was because the process already was irreversible The value of surgical sympathectomy has been proven in arterial occlusions and must be accepted It is particularly valuable early, prior to the onset of gangrene, and in certain instances gangrene can be limited by surgical sympathectomy. It is not to be considered as a cure of arterial occlusion nor can it be considered like an appendectomy for appendicitis In the last 100 sympathectomy operations for arterial occlusion, only 13 came to amputation If we eliminate 2, where sympathectomy and amputation were done at the same time, and 2 where the ganglia were not found pathologically, we have a corrected 9 per cent amputation rate The relative high percentage of diabetics who were amputated (see Table 12) reflects the part infection plays in the prognosis One dislikes to say to

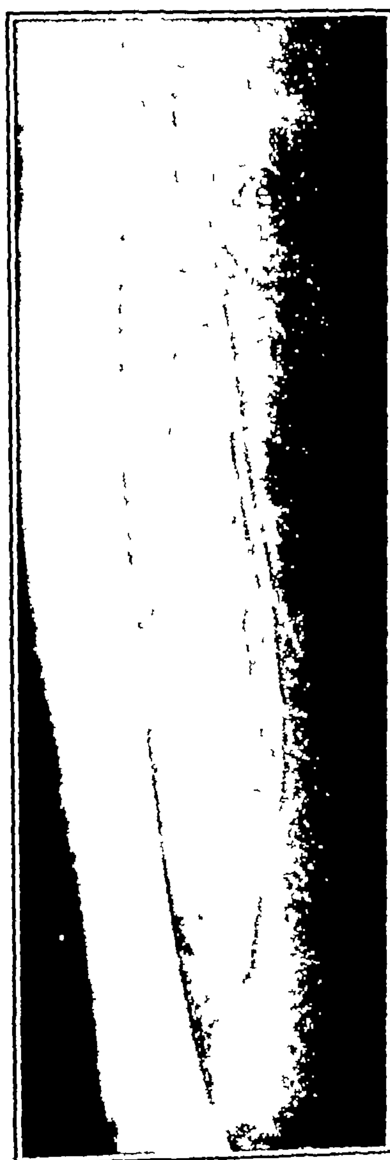


Fig. 13 Arteriogram showing segmental arterial occlusion favorable for by-pass operation This patient had arteriosclerosis at the age of 17 Vein graft performed

surgeons that the operation must be thoroughly done but in cases of failure the source may be the technic The sympathetic chain is elusive and difficult to identify for the inexperienced Pathologists report many substances submitted as sympathetic nerves and these include all types of nerves fat fascia and even ureters Our results vary directly with the completeness of the procedure and we no longer cut the chain but ablate it by an exeresis For technic see Interruption of Sympathetic System pages 487–525

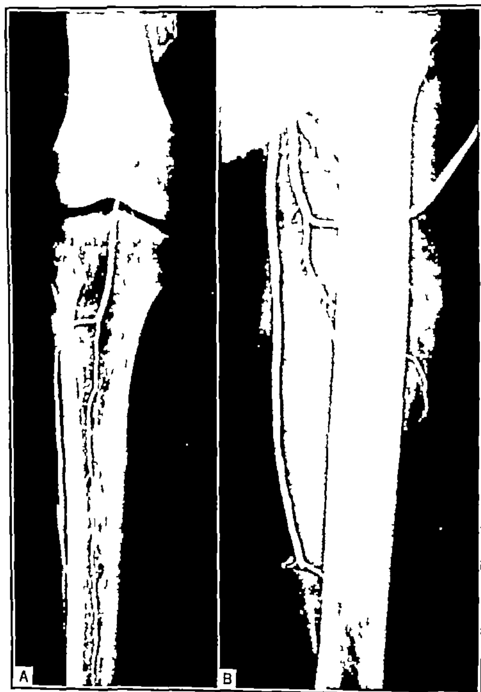



FIG 44 —Advanced arteriosclerosis with segmental occlusion. Vital detour artery well-shown. Treatment was endarterectomy

a

b

c

d

FIG 45 —End to side vein graft to bridge or by-pass segmental arterial occlusion. (*a*) Shows femoral artery occluded distal to the profunda—vein graft sutures in place Note collateral circulation of artery has not been interrupted *Technic* (*b*) Reversed vein split to make large opening and united by mattress sutures (*c*) Uniting vein graft with continuous suture (*d*) Similar closure with interrupted sutures (Author's angled needle facilitates sewing, see page 117 )



FIG 46 —End to side anastomosis of reversed vein attached above and below an occluded artery

(d) *Replacement Grafts* —Often in patients who have an arterial disease a thrombosis may occur in a segment of a vessel It may be possible especially if this is of the atheromatous type in which the rest of the vessel appears quite healthy to by pass the gap or thrombosed area with a graft taken from contiguous vein This is true most often in atherosclerosis It has been done using the saphenous vein femoral vein and/or the jugular vein at times It should be kept in mind and considered if

(1) the arteriogram shows a local arterial block

(2) the remaining vessel appears healthy and will hold suturing

(3) removal of the thrombus does not leave sufficient artery to conduct the arterial stream

(4) the collateral circulation is inadequate

(5) clinical evidence exists that there will be gangrene without this procedure

(6) the patient is young



Fig. 47 —Long vein graft from common femoral artery to popliteal artery after attachment.

*Saphenous or Femoral Vein Grafts* —To avoid the possible interference with the collateral circulation which occurs when a thrombosed area is excised a modification of the original vein graft technic has been used These vein grafts were suggested first by Leriche[41] and also performed by Kunlin[38] In this technic the saphenous or femoral vein is selected for the graft. With each branch transfixed the vein is anastomosed to the femoral artery above and below the block. Anastomosis is performed in an end to side manner with due regard for the valves and their affect upon the direction of flow This operation bridges the gap without destroying the collaterals It has been used sufficient times to determine its place in selected instances Its potentialities are great.

Bridging of defects due to disease now is more often feasible in obliterative diseases The sclerotic vessels sometimes however will not tolerate

sutures This method may be utilized in selected individuals, particularly in the younger age group, and the use of homologous arterial grafts or analogous vein implants should be kept in mind and utilized if possible The first work of this type was suggested by Goyanes[24] in Madrid in 1906 It was tried by Leriche in 1912[42] Kunlin has performed 31 vein grafts and 2 homografts[38]

The advantage of an analogous vein graft is apparent Some surgeons have resected the blocked areas and then bridged the gap by end to end

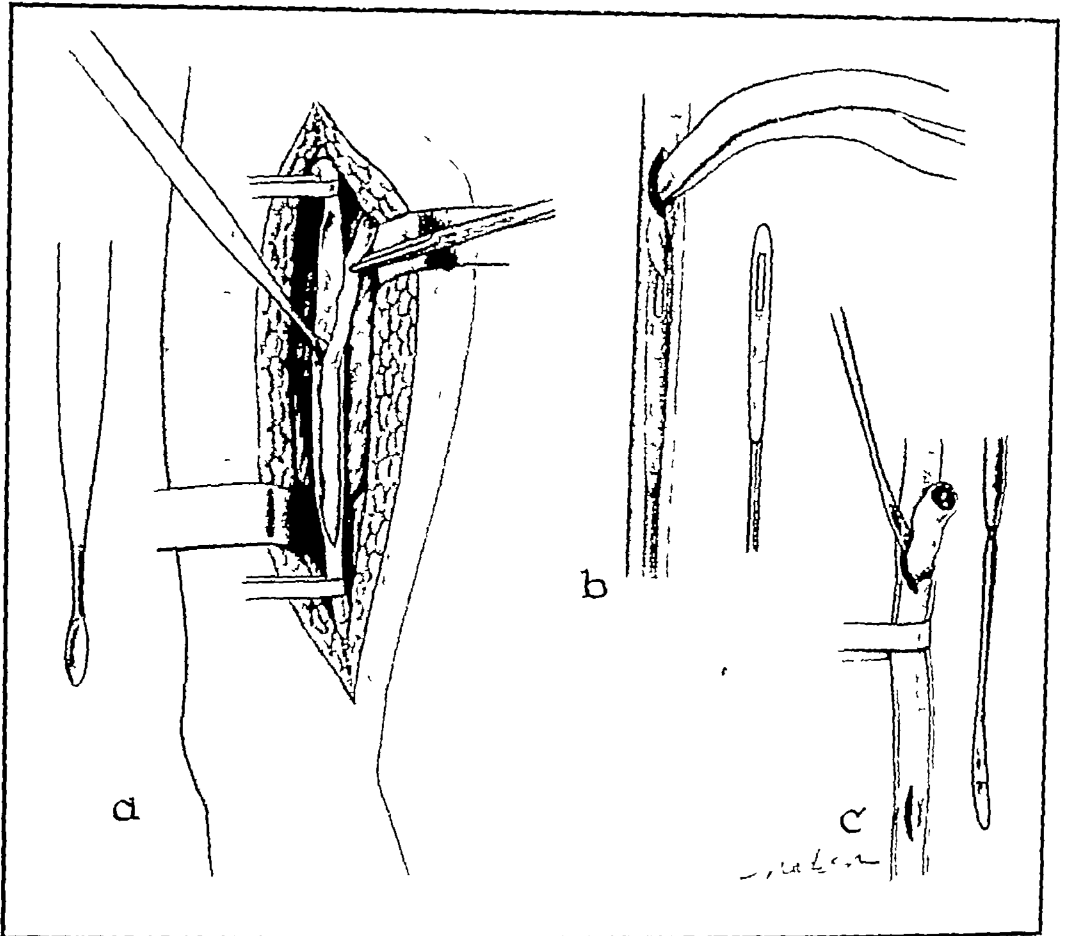



Fig 48 —Intimectomy, *endarterectomy and thrombectomy* *a* Artery has been opened, new and old thrombus, intima and media is removed with spatulas Adventitia then is closed with fine arterial silk *b* Methods of clearing artery debris where the occlusion is soft and unorganized It may be removed by vaselined gauze on the end of a probe similar to the cleaning of a rifle *c* Segmental removal of inner lining of artery, multiple small incisions Lining removed with spatula which is used to free thrombus at either end

anastomosis Such measures have the disadvantage of interrupting the collateral supply already present Following the work of Kunlin, we have utilized the long analogous vein graft We anastomosed these veins above and below the blocked artery after determining the site and extent of the block by arteriograms or aortograms The end of the vein is anastomosed onto the side of the artery The artery is opened, a window excised and the end of the vein sewed with continuous five-0 arterial silk Both interrupted everting and continuous over and over sutures have been used, but the latter

has proven the most satisfactory. The saphenous vein if large enough is an adequate bridge. If not, we use the femoral vein. In such instances this vein is removed without dividing any arterial branches to protect the already developed arterial collaterals. It is necessary to put the distal end of the vein onto the proximal end of the artery because of the venous valvular system. The vein is kept moistened with heparin-saline solution during the procedure. This direct attack on a blocked artery seems to be a real advance in surgical therapy. With more experience it may be the



FIG 40 —Excision of intima and media from artery

ultimate answer to the problem. The great advantage is that one does not interfere with the blood supply already present. One question still unanswered is whether the distal circulation can take away the additional blood load brought to it by such grafts. Also the question of future aneurysmal dilatation still is unanswered.

(e) *Intimectomy Thrombectomy Arterectomy and Endarterectomy* —In 1906 Delbet[13] suggested the re-establishment by surgery of an artery obliterated by arteriosclerosis. Many surgeons have discussed the problem but it was not until 1945 that Dos Santos [14] with the use of arteriography re-evaluated and stimulated thought on the reconstruction of the

obliterated artery The technical difficulty of the surgical procedure, the innumerable failures, and the difficulty of accurate arteriography did much to dampen the ardor of those early enthusiasts who followed Dos Santos' work[15] A paper by Bazy[3] before the American College of Surgeons accompanied by a motion picture showing the technic, has revived this work Therapy in this direction must be utilized and standardized to restore the circulation to a part—at least until such time as our medical and chemical research can work out the cause and prevention of athero- and arteriosclerosis

Failure of the earlier workers to achieve success may have been due to the fact that the anticoagulants were not available With the present day anticoagulants, and particularly heparin, much more can be accomplished



FIG 50 —Cleaning debris by "Vaselined" tape

Freeman[21] now inserts a segment of polythene tube directly above the site of the thrombectomy and drips heparin through it He has reported several successful outcomes after operations of this kind The thought that the only result of an arteriotomy would be the local interruption of the sympathetics to that area has been discarded It is true, however, that even if the circulation is not re-established through the previous obliteration, the site of the focus of afferent stimuli to cause the sympathetic reflex arteriospasm has been removed

*Technic* —Aortography or arteriography must be performed proximal to the site of obstruction The vessel and the site of occlusion is then studied as to degree of occlusion and the extent of the collateral vessels

*Technic of Intimectomy* —The area of thrombosis or of occlusion then is prepared surgically with a sulfonated detergent and the incision is made

under spinal or local anesthesia The artery is elevated into the field and an incision is made at the site of obstruction shown by the arteriogram The status of the artery is determined At times, the entire operation can be done through a small incision or several small incisions At other



Fig 51 Fig 52

Fig 51 and Fig 52 —Femoral arteriogram showing successful intimectomy Note the collateral artery at junction of the middle and lower third of the femur termed vital detour This artery opens when the femoral artery becomes occluded.

times the artery in its entire length of obstruction must be opened A point of cleavage is found from which one can separate the hard sclerotic internal cuff which has the calcium and cholesterol deposit in it from the outside cuff or covering of the artery The outside coat appears as a living structure while the inside one is a dead or sequestra like coating [3] This

dead part of the vessel consists of all but the adventitia A new vessel is then constructed by carefully suturing the external coats together with fine arterial silk There is no endothelial lining to this structure Clotting is prevented by the use of anticoagulants The inner lining soon endothelializes Prior to the time of endothelialization there may be a layer of leukocytes which lines the vessel and carries on the function of an endothelium until such time as nature has endothelialized this area [3] In the last years, increasing success has followed intimectomy Using small spatulas of the eye type, we have been able atraumatically to separate the thrombosed debris from the living external coat Where no true demarcation line exists we have opened also the artery distally at a point where the lumen is patent and arterial blood is returned from the distal end Various types of probes, strippers, and "ramrods" have been passed from one open end to the other and the debris between the two points literally removed as one would clean his shotgun With the development of better instruments this procedure can be extended to more patients

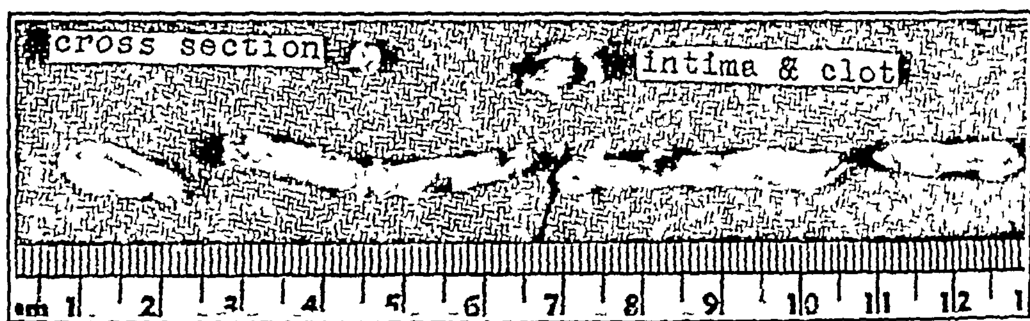



FIG 53 —Tissue removed from subject in Figure 51

*Indications for Intimectomy* —The indications for this operation are

1 Traumatic thrombosis in a normal limb

2 An acute area of thrombosis, particularly the undermining of a plaque in a diseased artery

3 A limb in which all efforts at conservative therapy are failing

4 If the patient has lost the other limb and is showing failing circulation in the opposite side

5 Thromboangiitis obliterans in the younger individuals who do not respond to conservative therapy

6 General arteriosclerosis in which some trauma has caused an area of thrombosis

7 If arteriogram or aortogram shows a localized obstruction

*Contraindications*

1 The absence of an arteriogram limits the use of this procedure

2 If obvious gangrene is present, the operation on the artery is not indicated

3 A spreading infection is a contraindication to any operation on the artery

4 Extensive involvement of the entire limb.

5 A thrombosis of the bifurcation of the aorta where resection of the aorta is probably indicated

*Conclusions* —The operation has an application whose limits are unknown at the present time It deserves consideration if other surgical measures and other types of therapy fail to improve the circulation status The development of better arteriography and aortography technic improves the operative possibilities Surgical sympathectomy should precede the operation



Fig. 54 —Demonstration of large collateral of femoral artery termed "vital detour" A vessel of this size is not present anatomically except when the main artery is occluded.

*Thrombectomy and/or Arterectomy* —In the patients where interruption of the circulation occurs from the undermining of a large plaque thrombectomy and/or resection of that plaque and that segment of artery can be done The diagnosis in these cases must be made clinically aided by aortography or arteriography The aortogram is made by the translumbar injection into the aorta and the injection of dye under pressure as described originally by Dos Santos,[14] visualizing the exact site of the obstruction If the clot is movable this thrombus is resected This must be done as early as one would do an embolectomy The circulation can be restored in a certain percentage of cases and residual pain can be relieved We have performed this procedure twenty-eight times with twenty-six surviving limbs

If the thrombus is fixed and not removable resection of the segment of the artery may forestall further clot propagations.

When thrombosis occurs at the bifurcation of the aorta, resection of the bifurcation is feasible in some instances This may be decided by the degree of calcification If the vessel is a mass of plaques, it is not feasible to try ligation or suturing and this procedure should not be attempted Sympathectomy should be performed with or without the resection In advanced cases like this, with thrombosis of the aorta at the bifurcation (the so-called Leriche syndrome), improvement at times can be expected



FIG 55 —Intimectomy iliac artery Lining of artery being removed with spatula Note preservation of all collateral arteries (silk threads around them)

No success has been achieved with thrombectomy in vessels distal to the popliteal artery Anticoagulant drugs are indicated after all such operations

(*f*) *Creation of Arteriovenous Fistulas* —The tremendous collateral circulation that is always present in the acquired or congenital arteriovenous fistula has impressed all surgeons Such a shunt stimulates Nature to make every effort to develop new vessels and branches to get circulation to the periphery The 2 to 4 vessels supposedly involved in the arteriovenous

process always are found to be innumerable vessels which must be ligated in the extirpation of the fistula The possibility of using this method to develop circulation was introduced by Bernheim in 1913.[4] We have now made three-way lateral fistulas between the superficial femoral artery and vein in 12 patients in each case reserving it for those in which it appeared gangrene was imminent Thus, the best candidates were not selected The fistula was made a quarter of an inch in size In 1 patient gangrene

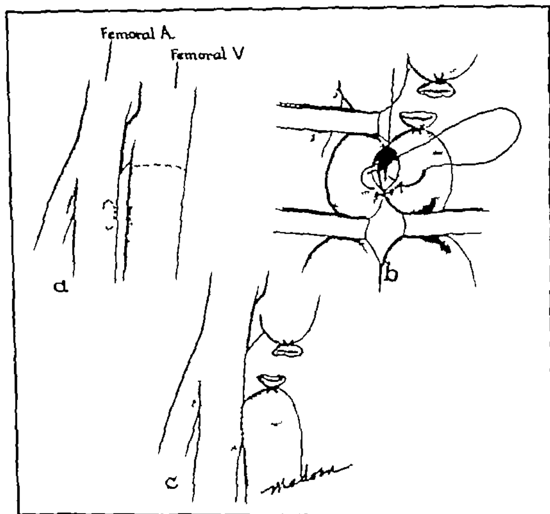



Fig 56 —Three-way arteriovenous fistula in an effort to stimulate collateral circulation Basis of therapy is development of the collateral circulation which follows traumatic fistula—experimental at this stage. (Pratt courtesy Angiology )

occurred at an accelerated rate In 4 no gangrene occurred In 3 gangrene occurred at the rate expected In 4 patients, the degree of calcification precluded a satisfactory anastomosis Winfield and Ruggiero [79] and Johnson [33] reported a salvage of from 12 to 20 per cent in their series with this type of therapy Whether it should be elected early still is debatable The fistula if constructed probably later should be closed

The work of Lillehei[41] and his associates at the University of Minnesota showed that if a fistula of sufficient size was created endocarditis

developed in 75 per cent of the animals so treated Since this therapeutic fistula is small, it is doubtful if it is of sufficient size to produce similar results The possible effect upon the heart and kidneys of such therapy, however, must be kept in mind

**Acute Thrombosis of the Aorta at its Bifurcation** (Leriche Syndrome) — All observers have noted the clinical entity of thrombosis at the bifurcation of the aorta, this closure sometimes coming on acutely Recently Leriche[40] called attention to this lesion as a separate syndrome

*Etiology* —Arteriosclerosis is the usual cause Rarely a thromboangiitis obliterans may be a factor Atherosclerosis, with the undermining of a plaque, may bring on the condition



Fig. 57 —Necrosis, slough and death of tissue from the bifurcation of the aorta distally following thrombosis at the bifurcation Multiple amputations failed to heal and surgical sympathectomy did not delay inevitable death Ante-mortem slough of buttocks and back

*Symptoms* —There are symptoms of an acute occlusion of the major supply of both of the lower extremities, and usually this is followed by rapid and often fatal gangrene There may be gangrene on the foot and this may extend rapidly Amputation at the site usually results in the failure to heal at the amputation site and many of these patients have been amputated repeatedly The end picture is one of spreading gangrene which involves all of the buttocks, thighs, and, as shown in Figure 57, the end result is fatality in most instances Figure 58 shows an aortogram of such a thrombosis The acuteness of the symptoms usually is due to the fact that the thrombosis occurs suddenly without the benefit of a collateral supply being developed

**Slow Thrombosis of the Aorta at Its Bifurcation** — Occasionally, thrombosis may occur quite slowly, and in some instances, the patient has been

able to survive without any treatment at all the thrombosis occurring so slowly that collateral circulation has been adequate. Ten cases of such slow thrombosis recently were reported by Elkin and Cooper.[18] Theis[78] also has a report on 25 cases. In 1 case of the author's there was no way to stop the process despite sympathectomy and bilateral amputation. Resection of the aorta was not carried out because of the patient's general precarious condition.



FIG 58.—Aortogram of thrombosis at the bifurcation of the aorta. (Courtesy of Dr B. Milanes, Havana, Cuba.)

*Pathology*.—The thrombosis which occurs at the bifurcation may be small or extensive. There are marked changes in the intima and at some times there are changes consistent with the diagnosis of Mönckeberg's[47] sclerosis. The final clot is usually a soft one depending upon the length of time that it has been present. The aorta may be calcified or riddled with calcium or cholesterol in any or all stages.

*Treatment*.—If the diagnosis can be made at an early date resection of the bifurcation of the aorta or thrombectomy or both may be feasible

In either case, sympathectomy should be performed In those cases in which the calcification is so extensive as to be likened to "a chicken's larynx," and this can be discovered by palpation at the operating table, it is obvious that the suturing of such an aorta at the proximal end will fail Sympathectomy alone, then, should be the treatment of choice The possibility of inserting either an inlaying vein graft or an aorta from an artery bank may be considered in such cases In these instances, if the vessel cannot be sutured, muscle may be packed between the inlying aorta and the patient's aorta and very lightly sutured Such surgery at the present time is in the "heroic" stage The mortality will be extremely high Involvement of the vessels to the kidney may be the determining factor Extensive and even fatal hemorrhage may occur if there is marked arteriosclerosis Bilateral sympathectomy, however, is indicated in each case Holman[29] believes that resection of the bifurcation of the aorta in general is too hazardous

*Debridement of the Vessel With or Without Vein Graft* —With the improvement in the aortogram and arteriogram technic, more of these patients can be surgically treated In the last years these x-rays have shown the segmental nature of the disease Many of these patients have been operated and the involved vessel opened, the bleeding controlled by rubber bands above and below the site We have been able to tease out the debris and thrombosis in some and to find a line of cleavage in others More recently we have opened the aorta at its bifurcation and then made a separate opening of the femoral artery Probes, strippers, and gauze have been passed through from one opening into another. The debris has been successfully removed in many patients. How permanent any improvement so obtained will be, still is unknown

The end to side anastomosis of an analogous vein graft will have some success in these patients The end of the vein may be anastomosed proximal to the block and distal to the obstructed area without resection of the obstructed artery This technic has the advantage of not interfering with the patient's collateral supply, as occurs when the resection of the segment is performed See pages 203 to 205 The vein may be reinforced by a nylon covering

**Peripheral Nerve Section** —Pain is such a predominant factor in some patients that despite the fact that gangrene is not present amputation has been done for this symptom alone It is not necessary to resort to amputation in all such patients Peripheral nerve resection of the involved sensory nerve may relieve this pain and save the patient's limb Resection is more effective than the chemical injections by novocain or alcohol The technic is not difficult Peripheral nerve resection has a limited application Such an operation removes the symptom of pain and also takes away a warning sign The complex superficial innervation of the foot adds to the possibility of error (See Fig 59 )

**Amputation.**—Amputation will become necessary in certain instances (see Chapter 15, p 234)

## DIABETES MELLITUS

Diabetes mellitus is a disease due to lack of internal secretion of the islets of the pancreas. As a result there is a sugar and fat dysmetabolism

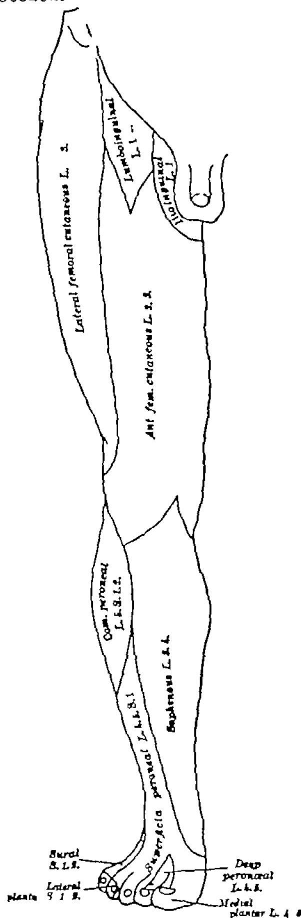



FIG. 59 —Complexity of cutaneous innervation of the foot. This explains why attempts at relief of pain by resection or injection of nerves often fails.

This dysfunction has been so well controlled that the patients live as long, or longer, than their nondiseased brothers The complications of the disease however remain serious Of these, the vascular complications are the most important The management of the diabetic necessarily requires a knowledge of vascular surgery The surgical consideration of diabetes mellitus divides itself into two types (1) the diabetic who requires a general surgical operation and requires special care because of the underlying diabetes, and (2) the treatment of surgical conditions to which the diabetic is particularly susceptible, such as arterial occlusion, carbuncles, and infections

TABLE 11 —INCIDENCE OF DIABETES MELLITUS—U S A

Increased incidence of admission to the hospital for diabetes and its complications

1910—3 per thousand, 1950—22 per thousand

| *Known Diabetics (all ages)* | *Undiagnosed* | *Potential Diabetics (all ages)* |
|---|---|---|
| 1,000,000 | 1,000,000+ | 3,500,000 |

*Total* 5,500,000—approximately 4% of population of United States

The number of known diabetics in this country, estimated by Joslin[84] to be over a million, is increasing due to earlier and better diagnosis and better management Through this excellent medical management, these patients live much longer than before and thus are alive at the time complications requiring vascular surgery develop

**Etiology** —Diabetes mellitus may be considered to be a disturbance of the pancreas with an inability of the body to metabolize adequately sugar and, secondarily, fat Other body organs secondarily are involved The condition of the liver is always a problem in the disease, and its status has to be considered surgically (See discussion on fat and cholesterol metabolism, pages 178–185 )

There are ten times as many admissions to large hospitals for diabetes mellitus today as there were in 1910 Forty years ago, for example, at Bellevue Hospital there were 2 8 diabetes mellitus admissions per 1000 individuals Now 2 per cent of all admissions are for diabetes or its complications Diabetics no longer die of their disease, provided their management is continued and complications are kept at a minimum The surgical aspects of diabetes are emphasized by the fact that 50 per cent of all diabetics require a major surgical operation during their life span

Insulin has minimized the danger in operating on the diabetic The safety of the present-day surgery of the diabetic needs emphasis Some physicians delay necessary surgical procedures because of diabetes No person should be allowed to die of one disease merely because he has another In diabetes mellitus this has occurred too often.

All types of surgery can be performed on the diabetic, including reconstructive, plastic or emergency surgery, provided the patient is prepared and kept under adequate diabetic control This control is best obtained by the coordinated action of the internist and the surgeon

Sometimes in the wards of city and general hospitals the internist's control ceases when the patient is transferred from medical to surgical service. A new and often unskilled regimen for management of the diabetic is instituted when the surgeon takes control. The care of such a patient should be a combined one with both the surgeon and the internist sharing responsibility.

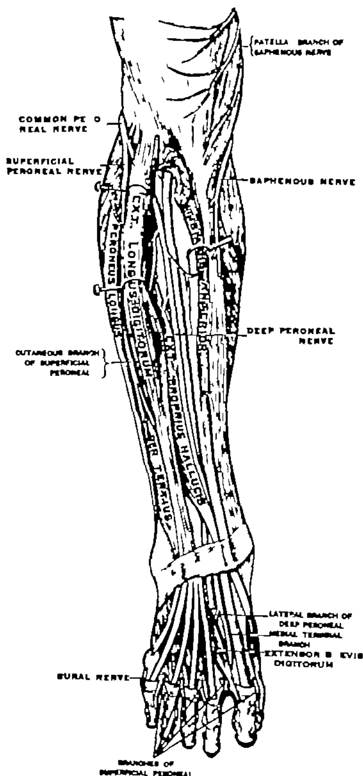



Fig 60.—Deep nerves of the front of the leg. (Gray's Anatomy.)

This community of thought is of value in diabetes mellitus, because despite any surgical complication that may arise, the fundamental condition will continue to be a medical one

**Surgical Operations on the Diabetic Patient.**—Operations on diabetics may be more dangerous than those performed on non-diabetics for the following reasons

Diabetic patients because of their disease are subject to

1 Metabolic imbalance
2 Infections
3 Acidosis
4 Liver dysfunction and disease

1 *A metabolic imbalance* may cause serious danger to the diabetic and is frequently present when the diabetic patient has a major operation The complications arising from such metabolism imbalance are.

*a* Vomiting
*b* Starvation
*c* Fever
*d* Excessive bowel elimination
*e* Cholesterol dysmetabolism

The part that a low fat cholesterol intake plays has been discussed on page 181

*Vomiting* is a frequent complication after an abdominal operation In a diabetic patient, this vomiting may so upset the glucose-insulin balance, both by the loss of food intake and the development of acidosis, that control of the diabetes is impossible The vomitus should be replaced by administering intravenous glucose Tests for acidosis must be run at regular intervals

A state of *starvation* is often present in diabetic patients with carcinoma or other wasting disease prior to operation and in the immediate days thereafter The enforced fasting depletes the necessary glycogen reserve in the liver and muscles with the result that nutritional supplements must be administered intravenously to replenish the lost stores

*Fever* is an important problem in these patients Fever depletes the glycogen reserve and at operation time the necessary sugar is not available The fluid loss also increases with fever and the fluid and protein reserve must be established prior to the operation in addition to the carbohydrate reserve

*Excessive elimination from the bowel* due to diarrhea, hyperperistalsis, suction-siphonage or where an ileostomy or colostomy is necessary may make the fluid loss so great that diabetic control is difficult The loss of salt and water is also a problem—potentially dangerous from a metabolic standpoint Again the need for the internist and the surgeon to work together is illustrated *A standard formula for diabetics at operation time would be that if the diabetic receives between 150 and 200 grams of sugar by mouth or by vein each twenty-four hours following an operation, together with sufficient insulin to utilize it, the danger of acidosis to that individual will be slight*

2 *Infections* and diabetes mellitus are incompatible The diabetic patient does not develop infection more often than the non-diabetic, but if an infection does occur, organisms multiply more rapidly and the diabetic control degenerates quickly We see this illustrated by their growth in sugar media used to culture microorganisms in the laboratory. The significance of infection is shown by the high amputation rate in diabetics

despite sympathectomy Seventy two per cent of the amputations were in the diabetic

Insulin also is less effective in the presence of an infection Infection may so rapidly direct the patient to an acidosis that his subsequent control is impossible

Prophylactic antibiotic therapy must be used The ability of certain antibiotics to reduce the number of microorganisms helps in the management of these conditions

For example sulfa drugs (Sulfathalidine, Sulfaguanidine and others) will decrease the number of Escherichia coli bacilli in the large bowel to such an extent that primary bowel resections now can be performed safely Prior to the advent of antibiotic therapy such a procedure was fraught with great danger Penicillin aureomycin chloromycetin, terramycin and streptomycin likewise have their place in this field The organism should be cultured and tested in the laboratory and the drug most effective against it should be employed prophylactically Most recent studies indicate that Neomycin and phthalylsulfathiazole (Sulfathalidine) given simultaneously are the best intestinal antiseptics now available[31]

Table 12. Amputation Incidence—100 Patients with Sympathectomy

| | | |
|---|---|---|
| Diabetes | (10% of Total) | 8 (72% of Amputations) |
| Arteriosclerosis | (90% of Total) | 3 (27.3% of Amputations) |

3 *Acidosis* may be confused with an infection or may hide its symptoms Typical symptoms of acidosis are *general abdominal pain tenderness vomiting and malaise* These symptoms are the same as those seen in an acute abdominal condition and one may mask the other

4. *Liver Changes* —Diabetics develop liver changes These changes interfere with the storage of glycogen cholesterol and also fat metabolism and affect the biliary functions and secretions As the diabetes mellitus advances these liver changes progress, making the control of the severe diabetic difficult. In patients who have a disease of the biliary system requiring a major operation liver damage also occurs When this is combined with diabetes mellitus it becomes even more difficult to control the diabetic condition An infection when present in the diabetic liver, is a very serious matter Biliary stasis also may be a serious complication in diabetic patients

**Diseases Incompatible with Diabetes Mellitus** —The diabetic patient is not only more susceptible to certain diseases but some of these diseases in combination with diabetes become even more serious The following diseases are incompatible with diabetes mellitus

1 *Gallbladder disease* has been found in 50 per cent of those who died of diabetes mellitus It is apparent, therefore, that diabetic patients who show signs of disease of the gallbladder should have this organ surgically removed early before the stage of biliary complications develops.

This point needs re-emphasis as frequently the patient with cholecystitis is treated medically despite the fact that he has a lesion amenable by surgery The development of complications therefore is inevitable, and the patient may have to be subjected to more drastic and dangerous surgery

than if he had been operated on earlier. The risk increases and the number who can be surgically cured decreases in direct proportion to the length of time that the disease has been present. In our series of elderly patients presenting gangrenous gallbladder disease, the mortality in those whose general state was so poor that only cholecystostomy could be performed was 50 per cent, while in those whose gallbladder was removed, the mortality was under 10 per cent. In addition, there was no death when the operation was performed before the sixth day of the disease. After the cholecystitis had been present for nine days, a septic peritonitis developed and death rate was 100 per cent.[90]

2. *Jaundice,* as a complication of biliary tract disease, markedly increases the mortality rate. It is believed that the jaundice interferes with the metabolism and storage of fat and glycogen.

TABLE 13.—SERIOUS INFECTIONS IN CLINIC AND NON-CLINIC PATIENTS (CARBUNCLES, BOILS, ABSCESSES)

| | |
|---|---|
| Non-clinic attendance | 21% |
| Clinic attendance | 0 |

3. *The pancreas* becomes diseased in the diabetic patient more frequently than in the non-diabetic. Of those patients dying in diabetic coma, one in every five shows an acute pancreatitis. Diabetic control is extremely difficult in patients with any lesion in the pancreas, and early medical and surgical treatment is advocated in such patients.

4. *Hyperthyroidism,* which increases metabolism, prevents control of the glycemia. The hyperactive thyroid has an adrenalin-like effect which is antagonistic to insulin action and probably also interferes with the storage of glycogen. Thyroidectomy will permit diabetic control in the patient who previously was impossible to manage. A small thyroid adenoma may be the cause of a diabetic patient's not responding properly to therapy. There should be no delay in performing the thyroidectomy, as later the two conditions both may be out of control and death may result. In this respect, radioactive iodine is a therapeutic aid. (See chapter on Radioactive Isotopes and Atomic Energy, p. 794.)

5. *Infections* are difficult to control in diabetics and this fact requires emphasis again. Boils, carbuncles, and infected ingrown nails are of dangerous importance in this group. The early mortality was from 20 to 60 per cent in carbuncle in the diabetic. With modern management and antibiotics, such serious states now are rare. Occasionally, however, a patient is susceptible to certain microbes and allergic or resistant to the drugs available for use. These exceptions present serious problems.

Treatment of these infections must be individualized but in general consists of active and aggressive diabetic management, antibiotic therapy, generally and occasionally locally, blood transfusions, and magnificent surgical restraint. The cholesterol-fat intake should be minimal and lipotrophic substances should be added. (See pages 178 to 185.) Joslin's[91] dictum: "Do not squeeze, do not pinch, do not cut, and do not run sugar," well summarizes the principles involved.

**Treatment of Infections in the Diabetic** —1 *Local Management* —Local applications of warm non-irritating solutions applied without trauma will aid They will keep the area soft localize the infection and will help splint the part A large piece of cotton over the area may be kept wet with a warm solution of saline or boric acid This may be applied with a dropper to prevent the trauma attendant to removing and replacing the cotton from time to time Palpation is prevented by this application If localization is complete and fluctuation is evident a simple incision and evacuation may reduce the time necessary to resolve the process The day of opening or excising a boil or carbuncle prior to its complete localization however has passed Such injudicious incisions destroy the defensive wall developed by the patient to surround the infection and may let loose an overwhelming number of microbes Immobilization of the part and of the patient is

TABLE 14 —DIABETIC INCIDENCE AND CLINIC ATTENDANCE (1045–1050)
(YEARLY AVERAGE OF 5 YEARS—ROUND FIGURES)

| | |
|---|---|
| Total Clinic Attendance | 125 000 |
| Total Diabetic Clinic | 1,800 |
| Total Diabetics in Vascular Clinic | 1 000 |

2% of all Clinic Attendance have diabetes

Greater attendance of diabetics in vascular clinic indicates importance of vascular complications.

imperative When the infection is on the face immobilization includes cessation of talking or at times even of eating intravenous or tube feeding being instituted These face infections are most dangerous as in this area there are no fascial spaces and limits to localize an infection The extreme elasticity of the skin and its immediate approximation to the muscles underneath it facilitates the spread of infection by movement. The angular vein which is without valves and in close proximity to the brain increases the danger of a complicating spread of the infection

2 *General Management* —*Antibiotic therapy* should be given early and must be adequate The drug most effective against the cultured organism should be selected and the susceptibility of the organism to the drug determined immediately in the laboratory Massive doses of the drug may be needed both generally and at times locally

The strength of the patient should be maintained by an adequate diet mineralization restoration of his vitamin level a lowered cholesterol-fat intake and complete diabetic control. *Small blood transfusions* at times may turn failure to success The transfusions are not given to replace hemoglobin or red blood cells With such an overwhelming infection the host must develop some deficient defensive factor in the blood if he is to overcome the infection In infection these elements are missing They can be supplied temporarily by blood transfusions The host destroys this blood in a few days and therefore another blood transfusion must be given every two or three days This supply of blood often is the determining factor in getting well even in the presence of adequate antibiotic therapy This applies whether the patient is a diabetic or not and was proven innumerable times to my knowledge in World War II

*Roentgen ray therapy* localizes some of these infections, and in selected instances may aid the outcome

The basic prophylactic measure, however, is elementary hygiene The patient who daily takes a thorough warm water and soap bath will rarely have infections of skin and hair follicles Brigham emphasized this fact with the terse statement· "The washed neck, like the watched pot, never boils " This applies to the whole body The detergent soaps with Hexacholorophene help prophylactically.

**Acute Abdominal Conditions in the Diabetic** —The diabetic will develop appendicitis, diverticulitis or other acute abdominal lesions as frequently as any other patient, but the differential diagnosis will be more difficult due to the metabolic imbalance The appendix, for example, ruptures frequently in the diabetic. A localized abdominal tenderness, persisting several hours despite adequate diabetic therapy, is an indication of some intra-abdominal lesion requiring explanation. In peritonitis following a ruptured viscus, diabetic control may never be obtained until the peritonitis is treated. In case of doubt, it is always safer to explore, and the surgeon who looks and sees instead of waits and sees will have the lower mortality.

TABLE 15 —CASES OF DIABETES MELLITUS PER 1000 (1952)

| *Male* | *Female* |
|---|---|
| 3 0 | 5 8 |

Surgical technic plays a part Large crushing clamps, operating with a scissors, mass ligatures and ties, and most of all a heavy hand, will cause not only a greater incidence of postoperative morbidity but will increase the mortality Asepsis of a meticulous type and careful hemostasis are essential. If the peritoneum is soiled by the stump of the appendix, an error usually not too serious in routine appendectomies, a fatal peritonitis may develop in the diabetic

**Carcinoma in the Diabetic** —The present longevity of the diabetic brings him into an age when carcinoma is encountered. Twice as many diabetics have operations for malignancy today as they did forty years ago. In addition, the diabetics are particularly susceptible to carcinoma of the pancreas, as illustrated by McKittrick's[85] and Joslin's[84] findings of carcinoma in the pancreas in one-third and one-half respectively of their patients who died of diabetes This disease must not delay operation for malignancy longer than the time nécessary for diabetic control

**Prognosis of Diabetics in Major Operations.**—The prognosis in the major operation on the diabetic will depend upon the patient's medical status. If the general condition of the diabetic is not as satisfactory as the nondiabetic of the same age, it is due to the fact that cardiovascular renal changes and degenerating arteriosclerosis occur earlier in diabetics If the degenerative changes are not advanced and the diabetic's management is adequate throughout the course of the operation, then the recovery period and the mortality should equal that of the nondiabetic whose cardiorenal vascular system is of equal status.

The diabetic should run a little sugar in the urine The cause of shock or coma or some anesthetic complication is better determined if the patient has hyperglycemia than when he is sugar free

Other technical points will improve the prognosis Despite contrary evidence advanced by certain clinics the diabetic stands a *short* operation better than a long one Administration of glucose and insulin during the operation improves the general status of the patient Early ambulation reduces the complications and should be part of every procedure including amputation Early ambulation is aided by nonabsorbable suture material Steel wire in our experience best fulfills the requirements of the ideal suture—that it be strong easily inserted and produce a negative or a minimal tissue response [29]

TABLE 10 —CAUSES OF DEATH

| *1900* | *1952* |
|---|---|
| Diabetes Mellitus ranked 27th | Diabetes Mellitus ranked 6th |
| Arteriosclerosis ranked 34th | Arteriosclerosis ranked 1st |

**Anesthesia in the Diabetic** (See chapter on Anesthesia p 27) —

1 *Local Anesthesia* —Local anesthesia is of value in operations in the abdomen neck, and head regions but it has shortcomings which must be understood The local injection causes skin tension and trauma Some stock preparations contain adrenalin a strong vasoconstricting substance Adrenalin should never be used in the diabetic both because of its local effect and because it will release sugar into the blood stream When there is an inadequate blood supply as in the extremities a local anesthetic should not be considered This applies also to the use of such solutions as ethyl chloride

2 *Inhalation Anesthesia* —Cyclopropane has proven to be the general anesthesia of choice Ether and chloroform should be avoided Nitrous oxide causes too great an anoxemia for routine use

3 *Spinal Anesthesia* —Spinal anesthesia is ideal It is rapid has a minimal general effect and permits early ambulation Spinal anesthesia temporarily performs a sympathectomy and if too high therefore causes hypotension This hypotension is a physiological effect and in the average individual unless extreme should not cause undue alarm Too vigorous efforts to counteract this hypotension with adrenalin or ephedrine is not physiological and may cause complications These drugs likewise may liberate sugar from the liver or muscles Our previous fear of using spinal anesthesia in the elderly patient no longer exists. The modern anesthesiologist can localize the spinal anesthesic effect For lower extremities anesthesia can be limited to the involved leg By eliminating barbitage and not overdiluting the anesthetic, its effect can be lengthened In the choice of an anesthetic agent one should choose the least toxic Procaine dissolved in the spinal fluid has worked ideally The choice of a hyper

or hypobaric solution further aids in the anesthetic effect Since the specific gravity of spinal fluid varies in different individuals and at different times, one must be aware of and counteract a sudden hypotension This variation is greater with some drugs than with others More toxic effects appear during extremely warm weather In the patient with advanced heart, kidney, or blood vessel disease, where hypotension may cause damage, the addition of drugs will counteract it

4 *Refrigeration Anesthesia* —Local refrigeration anesthesia in the past has saved many lives The technic of anesthesiology has so advanced that other agents, particularly spinal anesthesia, have replaced ice in nearly all instances In the moribund type of patient, or where diabetic control is impossible and when there is a question about the anesthesia technic, it is a useful and lifesaving agent Refrigeration to the anesthetic level is used at times where amputation is refused or must be delayed

Cooling may reduce infection Combined with a tourniquet it performs temporarily an amputation as it shuts off absorption from the part In extreme instances therefore, it may be used as an expedient to prepare a patient for amputation later It has been lifesaving

*General Refrigeration* —This subject is discussed on pages 34 to 36 The generalized hypothermia has a decided place in anesthesia for certain operations The metabolism may be reduced 15 per cent The circulation to the brain may be cut off for longer periods without cerebral damage by this method Certain precautions are necessary Blood loss at operation time, as in the aortic aneurysm, can be reduced

*Hypotensive Anesthesia* —This modality is used to reduce blood loss and add to the speed of operation The various methods of achieving a lowered pressure, the indications, and after-treatment are discussed on pages 34 to 37

**Diabetes and Peripheral Vascular Disease** —The process by which atheromatous and arteriosclerotic changes occur in the arteries is just beginning to be understood We know now that such changes occur many years earlier in the diabetic than they do in other patients The reason for the much earlier incidence of these changes in the diabetic is due to faulty fat metabolism (See pages 178 to 185 )

Joslin[84] believes that all diabetics develop sclerotic changes within five years after the development of the disease In young individuals this may be modified by adequate diabetic control

The incidence of cardiovascular complications in the diabetic will always be high At the present time, one-half of the diabetics die of some arteriosclerotic complication One may hypothesize that arteriosclerosis is a disease of faulty fat ingestion and metabolism and is due to failure of adequate sugar metabolism The reason it appears so early in the diabetic is because this sugar metabolism imbalance results in an early failure of fat synthesis

The diabetic with arteriosclerotic changes in his peripheral vessels becomes a permanent problem and can be compared to the patient with tuberculosis in that one may arrest or delay the process but probably never stop it This does not mean that diabetics must lose their limbs if they survive long enough, because, in our arrest of the process, collateral

circulation may be developed sufficiently to carry the load through noninvolved vessels as long as the patient lives Progress in revascularization, too is being made (See pages 108 to 210 ) The condition of peripheral arteriosclerosis, then is much the same as it is in the nondiabetic, with the exception that it appears earlier and when an infection develops its spread like infection elsewhere in the diabetic is rapid and sometimes uncontrollable

**Diagnosis** —The diagnosis is not difficult to make and no patient should be treated in any doctor s office today without a urinalysis and other indicated laboratory tests A glucose tolerance test should be a routine procedure When cholesterol-fat lipid phosphorous metabolism studies are available they will be most helpful Evidence of calcification will be present if the process has continued for a time The absent or restricted distal pulses rubor on dependency and pallor on elevation the history of claudication the difficulty of healing minor infections or ulcers on the feet together with the laboratory findings of urinalysis blood chemistry and the sugar tolerance test are significant

TABLE 17 —AMPUTATION INCIDENCE IN DIABETES MELLITUS (1942-1952)

| | |
|---|---|
| Incidence amputation per clinic visit | 0005 |
| Incidence amputation per hospital admission | 0025 |

**Symptoms** —The symptoms of arteriosclerosis or atherosclerosis in the diabetic are similar to those in the nondiabetic and have been discussed under Arteriosclerosis (p 186) The infection problem is frequently the factor that brings the patient to the physician Bone and tendon involvement occurs early A small infection around a toenail will develop into a cellulitis and lymphangitis shortly Claudication occurs with deprivation of adequate blood to the muscles. Trophic changes and loss of hair is seen The foot becomes cold shiny and at times atrophic The nails curl and *a fungus infection under them and between the toes is usual* The rubor on dependency and the pallor on elevation appear early Perforating and indolent ulcers are frequent complications

**Treatment of Diabetic Arterial Obliteration.**—The treatment has been detailed under Arteriosclerosis (See pages 188 to 210 and 234 to 254.) Briefly treatment divides itself into prophylactic measures and active therapy

**Prophylactic Measures** —Smoking must be discontinued completely The dangers of using nicotine and the hopelessness of attempting treatment if the patient continues smoking have already been detailed under the general therapy of occlusive arterial lesions This cannot be overemphasized It is even more of a problem in the diabetic than in the arteriosclerotic patient as nicotine has an adrenalin-stimulating action which releases glucose into the circulating blood

In general the prophylactic measures may be summarized as follows

1 Rigid diabetic control with reduced cholesterol-fat intake and adequate sugar with insulin to burn it to remove the endogenous and exogenous cholesterol products

2. Avoidance of any vasoconstrictor drugs, such as ergot or adrenalin, or nicotine Of these, nicotine is the most important

3 Hygienic care of the feet with precautions not to traumatize the limbs directly or indirectly to produce corns, callouses, burns, blisters, ingrown toenails, and to avoid irritating chemicals

4 Improvement and development of collateral circulation This includes sympathectomy

5 Use of lipotrophic substances (see pages 189 to 190)

6. Use of endocrine drugs, *i e*, thyroid extract and the androgen and estrogen hormones.

7 In selected instances where an arteriogram shows a local arterial block, a by-passing operation, an endarterectomy or arterectomy, may be used

8 In all patients who have shown loss of arterial supply to one limb or where a limb has been lost, prophylactic surgical sympathectomy on the other side should be considered

TABLE 18 —RELATIONS OF SURGICAL MORTALITY TO CLINIC ATTENDANCE

| | |
|---|---|
| Non-clinic attendance | 20% |
| Clinic attendance | 7% |

**Active Therapy.**—As in any arterial obliterative disease, the therapy depends on whether an infection is present and whether it is controlled When there is no infection or the infection is controllable, the surgical therapy emphatically is conservative The part is allowed to demarcate and, if necessary, to self-separate from the surrounding viable tissue During this stage, efforts are made to keep the area free of infection with antibiotic therapy, eliminate undermining, remove sloughs which have separated, and insure adequate diabetic control

1 *No Infection* —When there is no open lesion, treatment is as outlined under Arteriosclerosis, with the addition of all the points outlined under prophylactic treatment on the preceding pages Sympathetic interruption is of great value in these individuals and, as in the arteriosclerotic, should be done before the lesion is advanced This will improve the capillary bed circulation of the skin, help in the healing of some small necrotic areas, improve the claudication time in many, and may prolong the life of the limb and of the individual indefinitely

2 *Controlled Infection* —As before, all the prophylactic measures are instituted. When there is an infection, the therapy depends on its control As in the arteriosclerotic, if the infection responds, the necrotic area is treated conservatively Saline soaks, control of the fungus infection. Sitz baths, sterile dressings, and careful removal of sloughs, with the prevention of undermining, are important as they are in the arteriosclerotic

Necrotic toes are permitted to demarcate and, at times, are removed atraumatically with a rongeur, permitting the tendons to remain long to serve as drains Great care is taken of any ingrown toenail, corn or callous, and a carefully trained podiatrist who understands asepsis is consulted regularly

The action of the anticoagulants particularly in the stage where arterial occlusion is occurring has been stressed It has been proven experimentally that it requires a much lower freezing temperature to produce gangrene in the normal extremity when the patient is treated with anticoagulants By the same token a greater degree of spasm and arterial occlusion must be present to cause thrombosis and gangrene in such an extremity if it has been supplied with anticoagulant properties It is our practice to begin with heparin given in divided doses and also to give Tromexan or Dicumarol or one of the other oral drugs discontinuing the heparin when the prothrombin time has been lengthened therapeutically This technic is fully described in the chapter on Antithrombotic Substances page 651 The combination of whirlpool baths and a saponified detergent (pHisoHex) with Hexachlorophene has resolved many of these small infectious areas (See pages 170 to 171 )

3 *Spreading Infection* —If the infection becomes uncontrolled it will spread more rapidly in the diabetic than in the nondiabetic Insulin will become ineffective and diabetic control will be difficult or impossible to maintain

When the patient is seen for the first time with necrosis and infection present therapy is instituted in the form of antibiotic therapy soaks diabetic control and drainage of any localized lesion for twenty four to forty-eight hours If there is a subsidence or localization of the process such treatment is continued If there is progression of the process despite such methods amputation must be considered and not long delayed

(a) *Refrigeration and Non-Surgical Amputation* —In a few patients with an uncontrolled infection it has been possible to institute a refrigeration regimen thus decreasing the demand of the peripheral tissues for oxygen In some patients this technic is used with the application of a tourniquet above the gangrene or infection area This performs a bloodless amputation removing absorption from the limb With this procedure the temperature usually drops the diabetes responds to therapy and the entire picture of a moribund state changes simultaneously Unfortunately this does not always occur This therapy should be tried in desperate cases where the outlook otherwise is hopeless Amputation can be performed then electively when the general status has improved

(b) *Amputation* —There will be a time in some diabetics when amputation will be required These often are of a major type and at times must be through the thigh The amputation is performed to control the infection and the infection may be spreading Delay at such a time may hazard the patient s life When it appears that the limb may have to be removed the problem should be presented early to the patient and his family so that some psychic preparation can be made and the delay for consent minimized

Even with infection the diabetic foot can be treated conservatively until the infection progresses to the ankle The defense of the body above the ankle is poor and the infection may spread so rapidly that, if the amputation is not performed at once a septicemia may result

Antibiotic therapy at the time of operation is important This applies particularly to the gas gangrene organisms *Clostridium perfringens* (*Cl.*

*Welchii*). Gas gangrene antiserum, tetanus antitoxin, and the antibiotic to which the organism is susceptible should be given This is discussed in detail on page 457

The *amputation site* must be individualized. The selection of the site for amputation will be discussed in detail on pages 234 to 254 Some general rules however apply.

General principles:

A *Necrosis with Controlled Infection* —Where there is necrosis or ulceration, but infection is limited, these recommendations are made

1 If the lesion is limited to the toes, no amputation is indicated. These toes are allowed to demarcate, then to self-amputate, or the bone may be removed with a rongeur

2 If the necrosis is limited to the toes or surrounding areas and infection is under control, amputation may be performed through the foot The transmetatarsal amputation of McKittrick[85] may be used In our Clinic we have replaced this procedure with an amputation proximal to the metatarsals Sympathectomy should be performed as an added aid in most instances.

3 If the necrosis has spread to a point where healing is impossible but where infection is not marked, *amputation below the knee* is to be considered. This applies to younger patients.

If a sympathectomy has been performed with some response, amputation below the knee is to be considered

B *Necrosis With Gross Infection* —1 If the infection is limited to the toes without evidence of spread, amputation is not indicated Conservative therapy and antibiotic therapy should be continued

2. If the necrosis is limited to the foot and an infection is present but controlled, no amputation is necessary Local incision of any localized infection should be performed With antibiotic therapy and anticoagulant treatments, the process may subside If the condition does not improve in forty-eight hours but the infection is not spreading, amputation below the knee may be considered, particularly if the patient has had a sympathectomy

3 Where the infection or necrosis has spread over the dorsum of the foot and is not controlled, amputation is indicated and the site depends upon the age status and the vascular supply

**Prognosis.**—A program that combines hygiene, podiatry, diabetic management and vascular therapy will keep diabetic patients *with vascular disease* walking on their own limbs for many years Our amputation incidence over a five-year period is shown in Table 19

**Gangrene of the Heel.**—Gangrene of other parts of the foot besides the toes is not too unusual both in the arteriosclerotic and the diabetic, and particularly in the latter[67] Gangrene in this area may be precipitated by pressure In a foot in which the circulation already is precarious, even slight interference to the circulation by pressure may be the coup de grâce to the circulation of that part For example, a patient at bed rest may lie all day and night with the entire pressure of the foot directly on the heel and cause an ischemic area to develop This pressure is increased at one area by the use of such a so-called safety device as a rubber doughnut or

cloth window. This merely transfers all the pressure from directly on the heel to a ring or rim-like area around it. Patients in casts, extension apparatuses or bedridden from other causes particularly are susceptible.

*Treatment.*—With the development of such gangrene the part is not necessarily lost.

*Local Care.*—The area must be kept carefully clean. A sulfonated detergent such as pHisoHex with Hexachlorophene, benzine or ether can be used to cleanse the area and thus to help separate the devitalized fat. Bed rest is essential in the treatment of these conditions. A mild fungicide should be added such as 1:25,000 potassium permanganate solution used as a soak or zinc undecylenate (undecylenate acid 2 per cent, zinc undecylenate 20 per cent, talcum U.S.P., 78 per cent) in a powder or petroleum jelly base may help. Azochloramid in triacetin can be applied for its effect on some secondary invaders. Each day after an hour's saline soak, an effort should be made to separate the devitalized tissue from the living tissue. A localized infection should be drained and these soaks will help in its localization. Antibiotic therapy with the correct agent for the cultured organisms must be continued.

TABLE 10.—MAJOR AMPUTATIONS FOR VASCULAR DISEASES

| *Diseases* | *Number* | *Deaths* | *Per Cent* |
|---|---|---|---|
| Arteriosclerosis | 113 | 4 | 3 |
| Diabetes | 102 | 12 | 11 |
| Thromboangiitis obliterans | 20 | 0 | 0 |
| Total | 235 | 16 | 6.8 |
| Embolism | 19 | 8 | 42 |
| Total | 254 | 24 | 9.4 |

*General Care.*—An anesthetic type drug called Oranixon* (3-orthotoloxy 1,2 propanediol) (grams 1, 3 to 4 times a day) used in this stage to relieve pain has been found to be of value. Sympathetic nerve blocks may be of limited value. Surgical sympathectomy may resolve or limit the process. Vasodilating drugs in most of these patients have not been effective.

The control of infection determines the success or failure of this therapy. If the infection has subsided, further trials at debriding the area are continued. Lifting off the dead tissue below the pain or blood letting stage, is done each day. If there is bleeding or pain, this indicates that viable tissue is being disturbed and this is not good therapy.

*Local Applications.*—The greatest value of any local application is derived from the wet warmth of a nonirritating solution and the mild maceration that occurs from soaking the part for approximately one hour a day. As the slough is gradually removed, clean granulations will appear beneath the area if the therapy is successful. This requires the cooperation of the patient. This therapy cannot be done by an unskilled resident or interne as he may injure viable tissue and spread the infection or the necrosis. If the surgeon is willing to spend the time, a healed, clean lesion will

* Organon, Inc., Orange, New Jersey.

result more often than not, and during this time the patient can be trained in the importance of preventing similar lesions in the future The six or eight weeks necessary to heal this lesion are worthwhile and must be compared to the loss of an extremity for the patient Many surgeons do not try to save an extremity in this pregangrenous state, but from our experience, we cannot overemphasize the great value of the time and effort expended to the individual When one has reclaimed an extremity of this type for a doctor himself and sees the gratitude expressed from one who knows the alternative, he then realizes fully how much he is doing for his patients despite the time it takes This therapy should be continued even if there is hospital clamor for the bed space An individual with his two legs is worth so much more to himself and his community that this cannot be compared to the saving of hospital bed space The hospital days necessary for such therapy must be made available

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Chapter

# 15

# AMPUTATION INDICATIONS, TYPES AND TECHNIC

**Decision on Amputation.**—Surgical concepts of amputation in vascular disease have changed markedly in the last few years Most surgeons are concerned when they realize the number of legs which have been amputated in the past and many of these in the upper thigh Today such amputations might be prevented, or at least delayed, and certainly performed at a lower level Our present tendency is to be more conservative in all amputations as well as in the level of amputation The optimal amputation time varies in each individual The decision must be based on a consideration of many factors Of these the most important are infection and the vascular supply These two necessarily are related, and in turn depend upon age, neglect of an open lesion, response to therapy, the infectious organism and the cooperation of the patient In the past and in many institutions today, too many amputations are performed An amputation should be considered as a failure of all other therapeutic measures While such a statement is medically indefensible, patients have lost their limbs to resolve the process and make the bed available to some more active surgical lesion An over-zealous house staff or economy minded superintendent, at times, have swayed the decision

Where infection is minimal or controllable in patients with thromboangiitis obliterans and arteriosclerosis obliterans, even with diabetes, ultra-conservatism is advocated. A necrotic toe should be permitted to demarcate The dead end is removed with a rongeur, leaving the tendons long so that they can act as drains, thus not interfering with the epithelization which has been taking place at the wound edge while the part was demarcating If the infection spreads it becomes paramount and an amputation must be performed The ankle level acts as a natural defense against infection in these patients If the infection spreads beyond this area despite active antibiotic therapy and local care, amputation may be necessary as a lifesaving measure Whether such an amputation must be in the thigh or below the knee depends entirely upon the extent and rapidity of the spread of the infection With the chemotherapeutic agents and the antibiotics which are now available, one is able to control most of these infections, and the decision as to whether to amputate and the site can now be based in most cases, alone, upon the arterial supply to the part This premise includes even the diabetic patient, except in badly neglected instances

**Digit Amputation** —This amputation is rarely indicated It should be replaced by the self-demarcation process already discussed If an incision is made over a digit proximal necrosis is to be expected If it is utilized very loose approximation or no suturing is advocated

**Wedge Resection.**—Wedge resection of necrotic tissue and bone without suturing has a definite place in the therapy of these limbs The area should be left open, treated by sterile soaks and antibiotic therapy and permitted

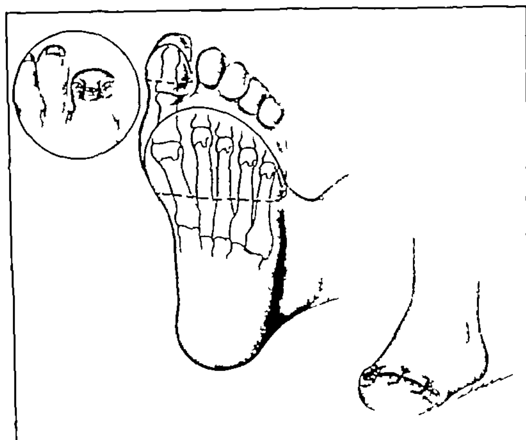


FIG. 61 —Transmetatarsal amputation. Amputation through the metatarsals (McKittrick Operation) This operation was developed in hopes of preventing necrosis after thrombosis of one of the digital vessels had occurred To be effective the operation must be performed early so healing will occur

SMALL INSERT Shows primary closure after amputation of toe where vascularity at operation time is found to be adequate Note that the skin is very lightly approximated with sufficient room between sutures for drainage

to fall together when clean The whirlpool bath to which is added the sulfonated detergent has aided in keeping the part draining and clean Figure 62 shows end results. Surgical sympathectomy is added to such resections routinely in our Clinic

**Metatarsal Amputation.**—With the increasing ability to control infection McKittrick [17] first in 1944 advocated an amputation through the metatarsal bones. His work was based on the belief that if one amputated through the metatarsal bones he could offer the patient protection against other digital artery thrombosis and subsequent involvement of the remain

ing toes and still have a useful weight-bearing foot The selection for patients for such an operation required that there be no spreading infection, that there be no infection near the amputation site and there not be the neurogenic changes as occur so often in the diabetic From this work came one valuable advance—the excision of an area of infection In certain instances it is possible to completely excise a necrotic area and get clean granulations at the site of the excision In others, of course, there may be failure to achieve such results

This type of amputation has not been too satisfactory in our experience It is difficult to rationalize the loss of all the toes of an individual only because one must be lost In addition such an amputation creates an open lesion which may not heal For such an amputation through the toes to




FIG 62 —Conservative management of occlusive arterial disease with gangrene *A* Loss of third toe and part of metatarsal *B* Loss of second toe and metatarsal. Patients able to walk satisfactorily for years

heal satisfactorily, it must be performed early in the disease, in other words, electively before infection is present and, in some instances, before it is apparent from the necrosis that the toe or toes must be amputated.

Many patients are walking with only one or two toes off and many others recovered without the loss of any toes These patients would have needlessly lost half of their foot if this early metatarsal amputation had been performed It must be remembered too that if the metatarsal operation fails, a below or above the knee amputation is necessary.

**Technic.**—*Preoperative Preparation* —Antibiotics should be used and the infection should be controlled until the part begins to demarcate. Fungus infection must be under control, and in our hands the use of potassium permanganate in a weak solution of 1·15,000 or 1:25,000 as a foot

soak twenty minutes every day helps In addition careful skin preparations should be made The G-11 preparations such as pHisoHex (with Hexachlorophene 3 per cent), is an invaluable aid in preparing this skin

*Anesthesia* —Spinal anesthesia is the anesthetic of choice in these patients.

*Technic* —The plantar flap is made long in order for there to be a firm solid base at the amputation site. The incision is made with a sharp knife, and sesamoid bones always are removed Undermining is avoided and careful hemostasis is achieved The wound is closed with fine alloy steel wire No 36 or 38 being adequate in our experience Under no circumstances should there be any skin tension or redundancy of the flaps

*Postoperative Care* —The patient's extremities should be kept slightly lower than the heart in order for gravity to aid the circulation in the part Motion of the part is continued and a modified type of Buerger exercise or the oscillating bed is begun at once If there is no systemic reaction, the wound is not dressed for at least seven to ten days Antibiotic therapy is continued Mobilization of the patient is permitted at once but weight-bearing is delayed until healing is effected Of McKittrick's[17] reported 215 patients there were 2 hospital deaths and 33 failures to heal There were an additional 39 in which there had been either a higher amputation or incomplete healing Primary healing was achieved therefore, in about one-third of all his patients. In our own experience we have used this operation in cases in which the patient has had an amputation of the other leg In those patients our figures of healing have run also about one-third of the patients operated Even combined with good conservative therapy it is likely that in no more than one-third to one-half of patients can healing be expected from this operation The operation therefore is advocated only for selected patients in whom it is hoped to prevent an amputation below the knee From our experience we would not advocate it as an elective procedure for those in whom only one or more toes are expected to be lost

**Tarsal Amputation.**—Tarsal amputation a modification of the Syme and Lisfranc operations proximal to the metarsal has been utilized satisfactorily This amputation has worked better for us than the McKittrick type In the latter the sharp metatarsals at times force their way through the skin. In the tarsal operation the plantar flap is loosely approximated to the dorsal one to allow drainage A specially constructed shoe with a stabilizing plate in the sole permits weight bearing The toe end of the shoe must be filled artificially

**Major Amputation.**—The preoperative preparation for a major amputation is similar whether the amputation is to be above or below the knee. The following statements on preparation and anesthesia apply both to thigh and calf amputation Similarly the postoperative care as to extension dressings ambulation and rehabilitation applies to both types of amputation

**Preoperative Preparation.**—When amputation is necessary every action involved should be made as simple and direct as possible in order to reduce the psychic shock experienced by most patients This can best be done by establishing a rigid routine and following it to the letter

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**Preoperative Preparation.**—When amputation is necessary every action involved should be made as simple and direct as possible in order to reduce the psychic shock experienced by most patients This can best be done by establishing a rigid routine and following it to the letter

The *general preparation of the patient* for amputation, like the preparation of the patient for any other type of operation, includes the emptying of the bowels with an enema and the fortification of the individual by vitamins, minerals and, if needed, the intravenous injection of glucose

Penicillin or other antibiotics and gas gangrene serum should be administered prophylactically after a skin test for sensitivity to the latter If the patient is dehydrated and lacks protein, these can be given by mouth or by intravenous injection The addition of 500 mg of vitamin B and 1000 mg of vitamin C to such injections will aid wound healing Chilling of the patient must be avoided

The *general preparation in the operating room* should be complete so that the amputation may proceed with machine-like precision The operating team, including the nursing staff, should be trained to work together and should be fully informed regarding the need for precision

To many patients a major amputation is like an execution—and like an execution, the quicker and less bunglesome the method, the easier it is to bear

Covering the patient's eyes, avoiding undue instrument and speech noises, and the preliminary inspection of everything that will be required for the operation will all reflect in reduced shock

The *local preparation of the operative site* involves a thorough cleansing of the limb with a G-11 preparation (we prefer pHisoHex which has 3 per cent Hexachlorophene in it) and the application of sterile drapes the day before the operation, again on the morning of the operation, and a third time in the operating room The first cleansing stimulates the sweat glands to secrete, and the second and third cleansings remove any infected sebaceous material that may have formed in between the scrubbings

Gas-producing bacilli* inhabit the colons of elderly individuals Since many of these patients requiring amputation are both bed-ridden and incontinent, the gas-producing organisms become lodged in the pores of the skin Repeated washings eliminate the source of infection entering through the skin

**Choice of Anesthesia.**—The choice of anesthesia for amputation has been considered briefly in the discussion of diabetes and anesthesia (see p 223) While refrigeration or ice anesthesia was considered ideal until recently, the development of newer technics in the science of anesthesia has made its employment unnecessary in most instances Its use was somewhat in protest to the increased danger and mortality that followed the employment of general anesthesias The previous bad results have been eliminated to a great extent Anesthesia is now safer and in hospitals where anesthesiologists of Board caliber are employed, the use of ice will be reserved for only an occasional patient The types of anesthesia that may be used will be discussed

1 *Spinal Anesthesia.*—This is an ideal anesthetic and has replaced all other anesthetics for amputations in our Clinic because of its efficiency, ease and demonstrated safety The time when the hypotension and shock, which may accompany such anesthesias, was a decisive factor has passed, and with the knowledge of the art of anesthesiology, these can be prevented. The anesthesia can be confined to only one lower extremity. At the time the bone is to be divided by a saw, one should protect the patient from hear-

* Clostridium welchii, Clostridium sporogenes, Clostridium oedematiens

ing this maneuver because of the possible psychic shock The anesthetist can place his fingers or plugs in the patient s ears for a few moments or divert the patient by discussing some problem Music in the operating room plays a part at this point

*Contraindications* —The only contraindications to spinal anesthesia would be

(*a*) Some disease of the spine

(*b*) Some physical reason making it impossible to be placed in the position necessary for spinal anesthesia to be given

(*c*) Inability to place the needle in the dura

(*d*) An extreme hypertension or hypotension making even mild variation in the blood pressure undesirable

(*e*) Some abnormal fear of the anesthesia which makes the patient entirely uncooperative

2 *General Anesthesia* —If a *general anesthetic* is required *cyclopropane* is satisfactory This anesthetic is a parasympathic stimulator and to some extent gives the effect of sympathectomy Cyclopropane has been used as an analgesic in patients who have appeared moribund The other gas anesthesias are employed rarely

3 *Local Anesthesia* —Local anesthesia while extremely safe is contraindicated in patients with vascular disease because the pressure following infiltration may constrict small vessels and cause local sloughing This is true particularly in the stock preparations containing adrenalin which is a strong vasoconstrictor

Local anesthesia should never be utilized in the foot itself Figure 63 shows the gangrene which followed the infiltration of a cuff of anesthesia around the base of the great toe for the incision and drainage of an ingrown toenail of a patient with thromboangiitis obliterans This photograph amply demonstrates that local anesthesia in the presence of arterial disease may cause gangrene

4 *Refrigeration* —Refrigeration as an anesthetic agent for amputation was first used by Allen and Crossman[4] who applied it to amputation surgery Fay s[9] work with ice on cancer patients was reported in 1941 In 1941 Allen[1,2] showed that by cooling with ice and ice water and by applying a tourniquet to the part of the limb distal to the tourniquet the area could be anesthetized and a comparatively bloodless shockless operation performed The author has reported on ice as an amputation anesthesia.[24] With this procedure many patients who are bad operative risks can be subjected to a major amputation and the mortality reduced

Historically it is of interest that Arbuthnot amputated legs using only a tourniquet to reduce the pain

The ice technic was used at first, only on patients who were in such a serious state that they could not stand any anesthesia. Today refrigeration is used for major amputations only if there are extremely valid reasons for not using spinal anesthesia. The patient s choice of anesthesia is not considered a valid reason for its selection

*Indications for Refrigeration Anesthesia* —(1) Severe advanced gangrene (2) Rapidly spreading infection in the lower extremity (3) Debilitated individuals who are unable to stand any other anesthesia (4) In severe

diabetes Even in these days an occasional patient will enter the hospital in uncontrolled diabetes with a high fever and gangrene, with uncontrollable infection The mortality always has been extremely high in this group The diabetes can not be controlled while the infection continues and if operation is performed at that stage death usually follows In these patients the limb can be elevated, put in ice and a tourniquet applied high in the thigh This tourniquet and ice stops absorption from the limb and the patient experiences the effect of the amputation Many times, the temperature drops and the diabetes can be controlled The amputation is then performed electively when the patient's general condition is better



Fig 63 —Gangrene of great toe which followed infiltration with local anesthesia for drainage of an ingrown toenail infection This patient had an undiagnosed thromboangiitis obliterans

*Technic of Ice Anesthesia* —The usual preoperative preparations are made. If an ice blanket is available, the part is wrapped in it three hours before the operation time If actual ice is to be used, the leg to be amputated is placed in the center of chipped ice so that the entire part is covered and ice is actually in contact with all of the skin from the groin distally Ice must be replaced from time to time The leg is elevated during the cooling Sedation is given at the beginning and during the refrigeration process The ice is not removed until the patient is on the operating table The final preparation is made with a G-11 preparation and cold sterile

water The amputation is then performed rapidly As soon as the main nerves are encountered they are locally blocked proximal to their division site with 1 per cent procaine The amputation is then completed in the usual manner the nerves being ligated and divided as the last step The stump may be kept cooled thereafter by the use of an ice blanket or an ice tent

The original technics included the application of a tourniquet This has not been found necessary in most patients

**Below the Knee Amputation.**—An amputation below the knee is always advocated if (1) the patient is under fifty five (2) the infection is minimal or controlled (3) an incision shows adequate blood supply at that level (4) a sympathectomy has been performed, (5) there has been an amputation of the other leg and (6) if a foot amputation has not healed

In making a decision to amputate below the knee many factors must be taken into consideration in addition to the age and weight-bearing of the individual A few elderly arteriosclerotic patients in addition to their gangrene are in a debilitated general status with systemic disease of the heart or kidneys While they may be able to withstand one major amputation if this failed and a second one were necessary it might be fatal The psychic shock of a second amputation is severe Many patients never recover their previous mental poise if it is necessary at a later date to reamputate above the knee

These statements regarding amputation sites pertain only to those patients who do not have an uncontrolled or overwhelming infection Where the infection cannot be controlled and gangrene sets in an amputation of course must be performed but only after the patient is adequately prepared with antibiotic therapy and the administration of gas gangrene antiserum (see page 457)

Except for the above deviations we perform the amputation below the knee in most patients At times this is combined with sympathectomy The extra fifteen or twenty minutes necessary to perform the lumbar sympathectomy in no way interferes with a successful amputation

A below the knee amputation is best performed with the patient lying on his abdomen. See Figure 64 showing anatomy

For an amputation stump to be satisfactory below the knee it should be in the mid tibial area The so-called elective site in the upper third of the leg preferred for the application of the old peg legs is not suitable for the modern artificial limb

A longer posterior skin flap should be made in the mid-tibial area to take advantage of the extra posterior calf tissue The optimal skin incision is made at a distance below the tibia tubercle corresponding to the space between the extended thumb and the little finger Soft tissues are divided 1 inch above the skin flap and the tibia resected 1 inch proximal to the incision site

The fibula must be divided at least 1 inch proximal to the tibial amputation level A Gigli saw or bone cutter is used to cut the fibula Operative procedures in which the fibula is removed entirely require additional dissection along fascial planes thus opening the fascial spaces to sepsis and increasing the operative time and trauma.

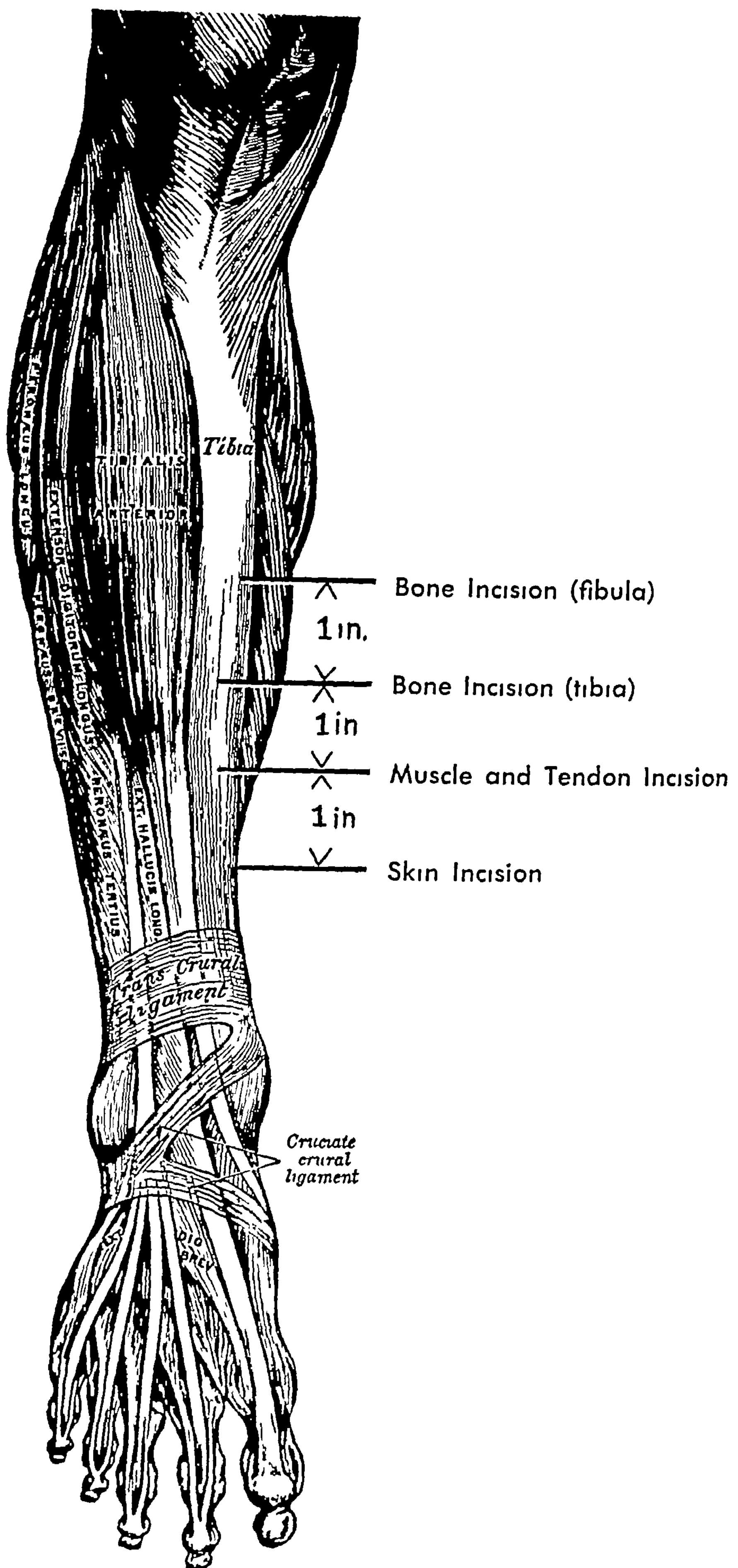



FIG. 61.—Shows that most of the soft tissues in the calf are posteriorly placed. The posterior flap is made longer than the anterior one and the operation is performed with the patient on his abdomen. (Gray's Anatomy.)

The anterior half of each bone should be beveled and all roughened edges removed with a rasp This procedure prevents the formation of osteophytes which, by their sharp edges, may be the cause of painful stumps The nerve is locally anesthetized and treated in the same way as described in the thigh amputation. (See page 246 )

As in the thigh amputation no effort is made to remove periosteum or tissue above the amputation site and no muscle or fascial flaps are prepared Hemostasis is secured

The wound is closed loosely with wire sutures and extension applied as described in the thigh amputation (See page 247 ) Particular attention must be directed to prevent the flexion contraction of the knee

The *postoperative care* of a patient with a below the knee amputation is the same as that detailed on page 248 for patients with a thigh amputation.

*Dressings* are not disturbed for ten to fourteen days unless there is an infection Antibiotics routinely are given to prevent any possible infection

An extension apparatus is maintained for at least three weeks and exercises are begun at once to prevent flexion contraction of the knee

As soon as possible the patient should be made to walk A rehabilitation program should be started immediately such as is discussed on pages 249 to 250

**Thigh Amputation.**—Before performing an amputation of the thigh the anatomy of the vessels and structures of the thigh should be reviewed

*Anatomy of Major Blood Vessels of Thigh* —The femoral artery begins behind the inguinal ligament It lies midway between the anterior superior spine of the ilium and the symphysis pubis

While anatomy books describe the femoral artery as being lateral to the femoral vein in the groin this is erroneous.

In nearly every instance the femoral artery is directly on top of or superficial to the femoral vein in this area The femoral artery vein and nerve are contained in the femoral sheath the so-called crural sheath which is a prolongation downward of the abdominal wall fascia the transversalis fascia being continued in front of the femoral vessels and the iliac fascia behind them This covering is strengthened in front by a band called the deep crural arch The sheath has compartments in it for the artery and the vein Some fibers of the psoas major part the femoral artery from the nerves in this area The femoral artery in the groin is immediately under the skin

As the femoral artery descends in the leg in the adductor canal it becomes more deeply situated and is covered by both the superficial and deep fascia and the sartorius muscle The femoral vein in this area then passes lateral to the artery The main branches of the femoral artery come off in the groin the perforating branches following the femoral profunda artery which comes off 1 to 2 inches below the inguinal ligament. The femoral profunda artery lies lateral and later medial to the main femoral artery

The circumflex and the other branches arise from the artery in the lower part of the adductor canal. These branches of the femoral profunda artery are (1) the lateral femoral circumflex artery (2) the medial femoral circumflex artery, (3) the perforating arteries including the genicular branch and (4) the muscular articular branch

In the popliteal space, the large vessels are in the posterior portion of the knee. The femoral artery comes through an opening in the adductor magnus muscle at the junction of the middle and lower thirds of the thigh, and follows down to the lower part of the popliteus muscle, where it divides into the anterior and posterior tibial arteries. This division may occur at a higher level. The femoral vein accompanies the artery.

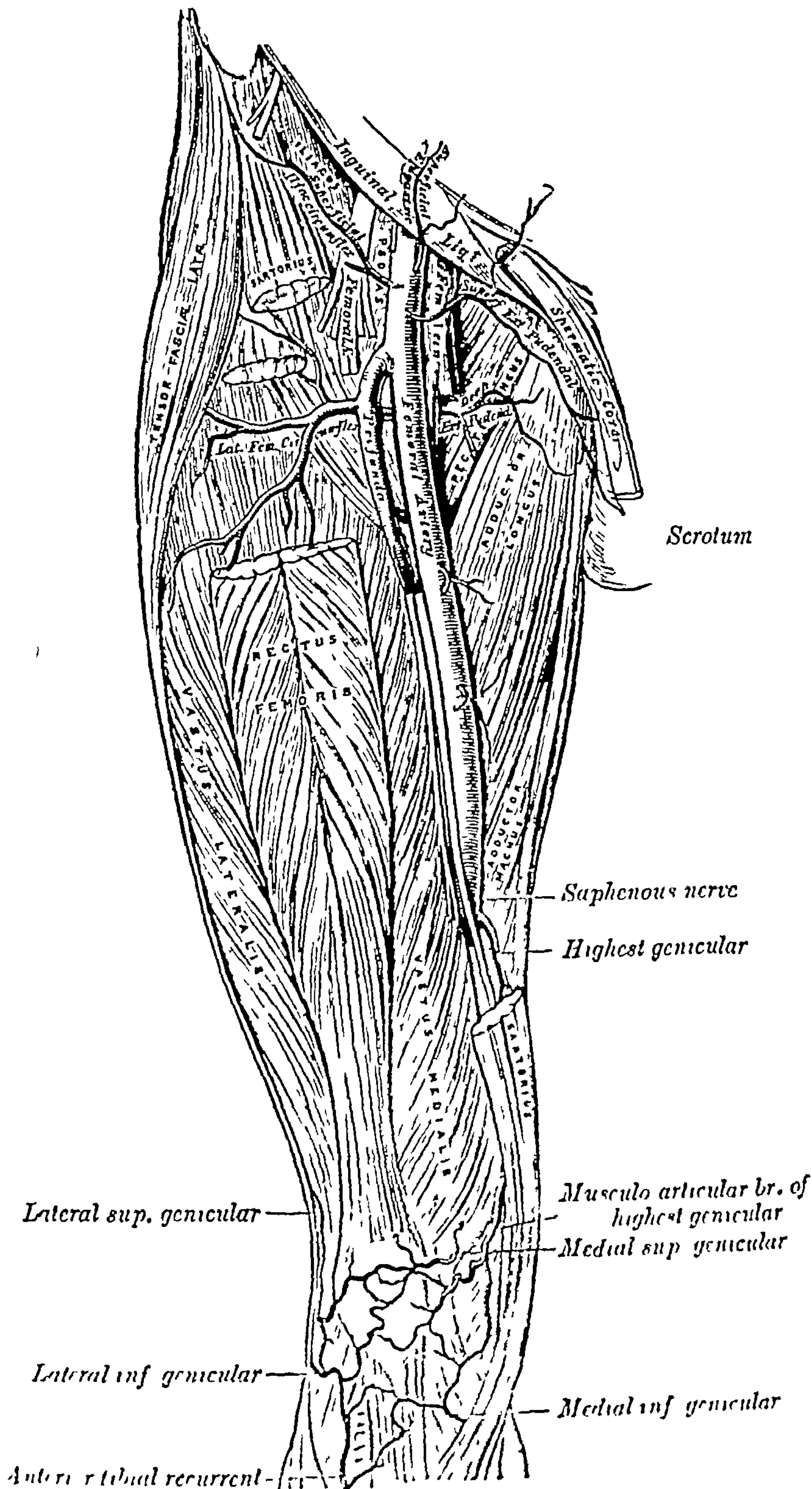



FIG 65 — Anatomy of the femoral artery (Gray's Anatomy) The femoral artery overlies the vein rather than lying lateral to it as shown in the picture

The artery in the popliteal space is separated from the femur only by fat tissue and the fascia covering the popliteus. Posteriorly, it is covered in part by the semimembranosus muscle and by the gastrocnemius and plantaris muscles below

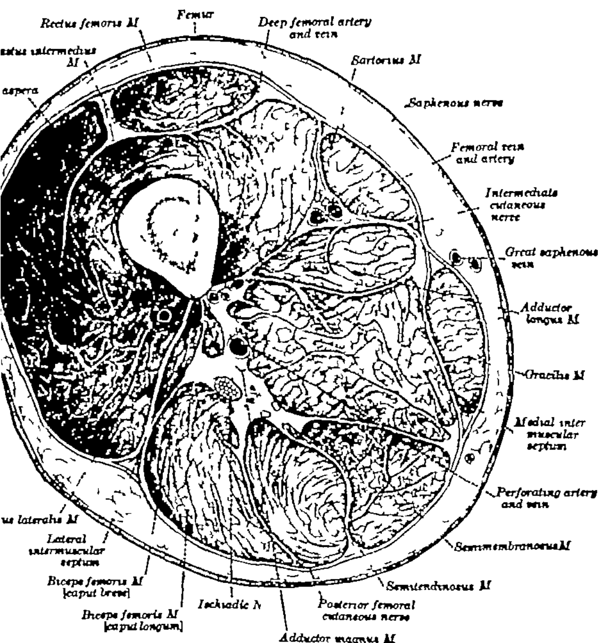



FIG 66.—Cross-section of the muscles of the thigh. (Gray's Anatomy) The level at which amputation is performed is lower where the muscles are more tendinous.

The femoral nerve which is the largest branch in the lumbar plexus arises from the second third and fourth lumbar nerves and its dorsal divisions. In the popliteal space the sciatic nerve is posterior to the major vessels. The sciatic nerve a continuation of the sacral plexus, is the largest nerve in the body. It is the first vital structure encountered in

opening the popliteal space from the posterior approach. With any growth or aneurysm involving the major vessels, the nerve will be brought more superficial and frequently will be flattened and stretched out.

These anatomical points should be kept in mind in performing any operation, particularly that of amputation

The indications for thigh amputation are.

1 Gas gangrene infection or other uncontrolled infections

2. Obliterative vascular disease in the aged in whom a secondary operation would be dangerous

3 Gangrene or infection in the lower leg

4 Patients where an incision below the knee shows avascularity

5 Trophic changes which would make the fitting of a prosthesis below the knee impossible

6 Patients in whom walking is never expected, *i.e*, cardiacs, one leg amputees, senile or psychotic patients

*Author's Technic* —The so-called plastic amputations have no place in the treatment of obliterative vascular disease Speed, the prevention of infection, the avoidance of undue trauma and shock, and the prevention of anesthetic complications are the principal aims of the surgeon

A circular incision is made through the skin at the level of the patella This incision is then completed posteriorly, but 1 inch higher. The muscle groups are divided 1½ inches above the edge of the upper skin flaps, at a point where they are tendinous

The large vessels are clamped and divided early in the medial posterior aspect of the thigh

The sciatic nerve is pulled down, its proximal end injected high with 2 per cent novocain and its division delayed until all other structures are severed We have seen a severe shock ensue from the unguarded division of this largest nerve in the body (See Fig 66 [cross section of thigh])

The soft tissues then are retracted a distance of 2 inches and the femur amputated without making a periosteal incision The soft tissues are not separated from the bone above the point of its division As the use of a prosthesis depends upon the pressure on the side of the thigh and not on the stump end, it is not necessary that there be a muscle or fascial padding over the end of the bone

A Gigli or hand saw is used in the bone division All rough edges on the femur should be smoothed with a file or rasp Bleeding is then controlled

*Treatment of Sciatic Nerve* —Our treatment of the large nerve is merely to ligate it with a fine steel wire and divide it on tension with a sharp scalpel and permit its retraction Alcohol injections and plastic procedures on the nerve result in more nerve symptoms than this simple method and in addition, at times causes wound sloughs and delay in healing Pathologic sections showed less tendency to neuroma formation after the treatment of the nerve by simple ligation [10]

*Closing the Thigh Wound* —The surgeon performing amputations for vascular disease is advised strongly not to place muscle and fascial ends over the severed bone The danger of protrusion of the bone end can be avoided by an extension applied to the skin as is described Flaps make pockets and pockets produce pus Extension and no flaps eliminate this complication

It has been known since the amputations performed in the war between the States that flaps over the bone end are unnecessary and undesirable Contemporary medical textbooks and articles nevertheless continue to describe methods of making fascial and muscle flaps across the bone end The wound is closed with three or four interrupted wire sutures placed through the skin and superficial fascia only The wire causes the least tissue reaction in our experience and may be left in place for an indefinite time Drainage can occur between the wires The wound is dressed and bandaged and an extension stockinette is applied This is applied to the skin by dermatome or adherent glue Two to four pounds of weight is attached to the end of the stockinette to provide the extension

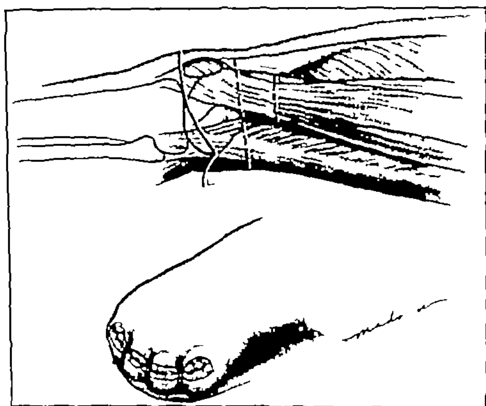


Fig 67 —Levels at which the skin the muscles and bone are divided The wound is closed simply with a few interrupted steel wire sutures in the superficial fascia and skin only

**Postoperative Pain** – *Phantom limb* is a normal postoperative symptom and in time disappears. *Phantom limb pain* is an abnormal condition In some patients these pains are of psychic origin but in others they are traceable to the presence of a neuroma The part that the psychic factor plays is definite

One group of 12 amputees evacuated in World War II all complained bitterly of phantom limb pain Careful search showed that one of the patients had a brother who had had a major amputation and had complained of phantom limb pain He described it to the others and thus caused the widespread complaint. This was the exception and not the rule in amputations seen in the Naval Service during World War II

Other sources for the pain must be checked A patient with phantom limb pain after an amputation of an upper extremity was also a chronic alcoholic and resisted any therapy for the alcoholism until the phantom limb pain was relieved Multiple nerve injections, four plastic operations on this nerve, and, later, a sympathectomy did not relieve the phantom pain This patient is an example of a psychiatric problem His "pain" was a defense for using alcohol and refusing to try an alcohol cure Definite neuromatas, however, need surgical care



Fig 68 —Photomicrograph showing lack of reaction of tissues to steel wire This wire had been inserted four years previously × 45 (Pratt, courtesy of Surg, Gynec and Obst )

**Postoperative Care.**—Postoperative care after an above or below the knee amputation, in general, is similar This applies to the extension, routine for dressings, early ambulation, and rehabilitation

1. *Extension* —The extension apparatus not only prevents skin retraction, but maintains the immobilization of the wound end necessary for healing and helps to obliterate the dead spaces by its pressure It is not removed for fourteen days Where there is excessive drainage, additional weights on the extension will reduce it Slight blood oozing may be decreased in a similar fashion Extension does not interfere with early ambulation

2 *Dressings* - The wound is not dressed for ten to fourteen days after the operation, unless there is a systemic reaction One should assume that the wound is surgically clean when he finishes the operation. There will be some drainage which can come out through the loosely inserted

wire sutures Redressing these wounds predisposes to contamination and infection as an ideal media exists for sepsis This non-dressing technic is responsible in part for lowering our morbidity from 75 per cent to under 10 per cent Primary unions ensue most often when the wound is not disturbed

3 *Ambulation* —The patient should be out of bed the day of operation and try walking in a walker and then crutches may be used after a few days The provision of a prosthesis can be attempted after six to twelve weeks Prior to the fitting of the artificial cuff the stump should be shrunk for a month by means of bandaging and soaking in a brine solution

4 *Rehabilitation* —Rehabilitation is most important and should not be delayed The cooperation of the patient s family employer internist and minister rabbi or priest should be enlisted See below A mental and

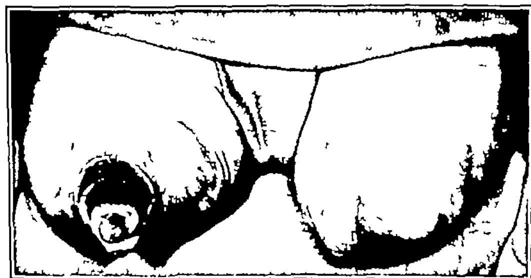


FIG 69 —Protrusion of femur through amputation stump due to failure to continue extension long enough. Re-amputation required increasing the risk.

physical preparation of the patient for the artificial limb is most important.

This long neglected phase in surgery has been emphasized by Rusk and Sverdlik [26] Most peace-time amputees being elderly physically and psychically shocked by the procedure and the victims of home sympathy were relegated to a sedentary life and usually died in a short time In t armed services where the individuals were young the necessity for rehabilitation program was recognized early and the exclusion of symj thetic families made its success much easier

The carry-over of this program into civilian life has made one of t great changes in the postoperative handling of the amputee

If there is time the problem should be frankly discussed with all co cerned Exercises to strengthen the shoulders arms and hands shou be begun

The greatest fear of any individual is that he will become a dependent. The patient should be assured that he is *not* going to be helpless, and motion pictures and discussions with a walking amputee will help to convince him

After the operation, the patient should be out of bed and in a walker, unless there is some cardiac reason to prevent it He should be encouraged to go to the bathroom, and to make his own toilet by himself at once

The crutches should be carefully measured and the patient taught to use them early

It is important for those interested in this problem to be kind but firm, to understand that every amputee wants to be able to care for himself but has great fear of his inability to act, and most of all, of riducule

Group drills, whether on a ward, semi-private or private status, should be encouraged, for the patients like to compare their progress, and the difficulties of some encourage the others The early depression and shame over their helplessness can be replaced by enthusiasm engendered by this group training

The rehabilitation program should include active work If the amputee is unfitted to pursue his former occupation, he should be vocationally trained in a new job while he is being restored to health

Only when the individual is fully and gainfully employed in a job he likes and is capable of getting around satisfactorily and with ease, can his rehabilitation be considered complete and successful

**Gas Gangrene Infection.**—During the second World War, it was found that the incidence of gas gangrene infection could be limited by the administration of penicillin in massive doses Gas gangrene serum is indicated prophylactically and therapeutically If there is any indication of infection, the dosage should be increased, and massive doses administered

Six seriously wounded patients with gas gangrene received aboard a Naval Hospital Ship in World War II were controlled without an amputation or death by open drainage and injection of six million units of penicillin daily

One must differentiate between the mere presence of gas gangrene bacillus and gas gangrene infection *Clostridium perfringens* (*Cl welchii*) may be a contaminant in the wound without causing the infection

*Symptoms of Gas Gangrene Infection* —Gas gangrene infection is characterized not only by the presence of *Cl perfringens* in the wound but by a high fever, rapid pulse, anxious appearance, and frequently, disorientation Gas is present locally in the subcutaneous tissues, and there is a sanguinous-like discharge A positive culture of the organisms from the wound, while disturbing to the surgeon, need not require re-amputation unless the symptoms of the infection are also present In World War II in one theatre, an overzealous medical officer on an evacuation ship performed five amputations because he cultured the gas gangrene bacillus from the wounds of his patients I had observed these same patients for a week previously and was certain, clinically, that they did not have the disease

While one cannot procrastinate in the presence of actual gas gangrene infection, a positive culture of the organism alone does not mean that there is an active gas gangrene infection unless the symptoms and signs of the disease are present

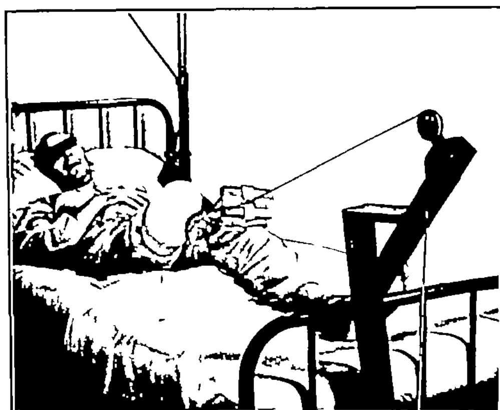




Fig. 70.—Exercises and early ambulation after amputation (Pratt, courtesy Postgraduate Med.)

The general and local symptoms of gas gangrene infection are summarized below

| *General Symptoms* | *Local Signs* |
|---|---|
| a Exposure to infection | a Sero-sanguinous drainage, early |
| b High fever | b Swelling |
| c Toxemia | c Pain and tenderness |
| d Disorientation and delirium | d Crepitation, later gross evidence of gas |
| e Marked tachycardia, leukocytosis | e Roentgen ray evidence of gas |
| | f Positive culture |

**Amputation Mortality.**—Amputation mortality rates throughout the world are extremely high[8,12,13,15] A review of the literature indicates a mortality in major amputations for vascular disease ranging from 20 to 50 per cent with morbidity reaching a figure as high as 85 per cent

The average of twelve New York City Hospitals showed that from 1930 to 1945 there was a mortality after thigh amputations for gangrene in the diabetic of 38 8 per cent The mortality statistics in other cities varied from 11 to 56 per cent

Mortality reduction is dependent upon

*a* Control of the infection
*b* Conservative management if control is effective
*c* Effective diabetic control
*d* No procrastination once amputation is inevitable
*e* Early and complete sympathectomy

TABLE 20 —MORTALITY IN MAJOR OPERATIONS FOR VASCULAR DISEASES

| *Diseases* | *Number* | *Deaths* | *Per Cent* |
|---|---|---|---|
| Arteriosclerosis | 113 | 4 | 3 |
| Diabetes | 102 | 12 | 11 |
| Thromboangiitis obliterans | 20 | 0 | 0 |
| Total | 235 | 16 | 6 8 |
| Embolism | 19 | 8 | 42 |
| Total | 254 | 24 | 9 4 |

Our 9 plus per cent mortality rate compares favorably with others[27] throughout the country McKittrick's[17 18] mortality figures remain excellent

The low mortality in our series is due, we believe, to controlled spinal anesthesia, a combined medical and surgical service, a careful preoperative preparation, a simple operative technic without flaps, ambulation thereafter, and not disturbing the wound after the operation The part that chemotherapy plays cannot be overemphasized

Since adopting the technic described, our ten-year mortality in major amputations has been 10 per cent, and only 1 patient has died in the last 54 amputations The wound infections, which we previously expected, have disappeared and primary healing occurred in over 85 per cent of the cases

**Summary of Surgical Management of Obliterative Vascular Disease** —

1 Conservative therapy in obliterative vascular diseases should be continued as long as avascularity is the prime point and infection is absent

or controlled When infection becomes uncontrolled more radical measures are indicated and a major amputation usually is necessary

2 Sympathectomy should play a larger part in this conservative surgical therapy The value of sympathectomy is shown by the fact that in the last 100 sympathectomies for obliterative arterial disease only 0 came to major amputation

3 Local toe self-amputations wedge resections, transmetatarsal or pre-transmetatarsal resections are applicable in selected patients

4 In the obliterative vascular disease associated with diabetes mellitus infection is frequently a complication and conservative therapy only is applicable in controlled patients An optimum amputation time can be obtained only once and if this time is passed danger to the life of the individual will result When amputation is necessary in the diabetic it should not be delayed

5 For amputation careful preoperative preparation is important emphasizing besides the general preparations a thorough skin cleansing (G 11) administration of antibiotics and gas gangrene serum prophylactically and the avoidance of chilling

6 Spinal anesthesia has proven the most satisfactory one from a mortality standpoint

7 A simplified amputation technic without the construction of muscle or fascial flaps or other plastic measures has resulted in a high percentage of successful operations in both above or below the knee amputations

8 An extension apparatus to prevent skin retraction of the soft tissue should be applied and maintained

9 The avoidance of fads and innovations based on a few cases will be reflected in the lowered mortality and morbidity

10 Steel wire is an innocuous suture and in our experience causes less tissue reaction than other suture material

11 Amputation wounds should not be disturbed for ten to twelve days unless there is untoward systemic reaction

12 Early ambulation reduces mortality and morbidity

13 Medical treatment of the underlying vascular disease must be continued from the minute the patient returns from the operating room or lesions may result elsewhere

14 A rehabilitation program should be part of the surgeon s responsibility

**Prosthesis.**—A difficult problem is presented to the surgeon when the patient asks him what type of an artificial limb he should obtain The question has no satisfactory answer To the patient however, the problem is one which he believes his surgeon can solve for him There has been very little progress made in the development of artificial limbs since peg legs were discarded Modern efforts toward the suction socket were stimulated by the World War II amputees. It is likely that this limb when perfected will be the answer to most of the problems Other than the suction socket limb most limbs available today are of standard design Whether they are of wood or metal construction, is an individual selection problem Many patients like the feel of a limb about the same weight as their own leg Others feel better with the light aluminum type

Some patients, particularly the young, are able to ambulate with limbs with such features as a mobile ankle joint Such a limb can adapt itself when placed upon an uneven object, such as a stone Other modifications include a locked knee on extension It has been our practice to specify at least three responsible limb manufacturers and let the patient "shop" for the one he likes the best Fortunately, most limb salesmen are amputees themselves and are able to demonstrate and extol the virtues of the various prostheses

*Suction Socket.*[5]—The suction socket depends upon an accurate fit The stump usually hypertrophies in such a limb in contrast to the atrophy which follows the use of most belt-type limbs This stump, then, requires additional refits Its use requires much cooperation from the patient. The amputation stump for above the knee suction sockets must be from 3 to 4 inches above the adductor tubercle to provide the necessary air space upon which the successful fit depends Exercise, prevention of hip flexion contracture, and wrapping of the stump prepare the limb for the suction socket The most important part is a qualified limb fitter The pressure within the limb varies with weight bearing Severe muscular contraction may so vary this pressure as to make the use of this limb impractical in certain individuals The patient must be of the composed mental type to acclimate himself to the use of this limb He will need encouragement

The fact that amputees have taken part in athletics should be stressed Major League baseball games have been won by a pitcher with one leg, and tennis champions, skiers, and even acrobats have become so proficient in the use of the limb as to be outstanding in their chosen sport

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Chapter

# 16

## ANGIOSPASTIC DISEASES AND SYNDROMES

In many diseases of the vascular system, both of organic and functional origin, spasm plays an important and, at times, decisive part. A classification of these syndromes is difficult since the spastic part may be temporary or only a portion of the pathologic picture. These lesions vary widely in their symptoms and include such diverse lesions as the excess perspiration of hyperhidrosis and the changes due to exposure to cold. They all have one common denominator, either in their origin or at some stage in the disease, and that is the spasm of an artery or segment of an artery in response to some stimulus which may be physical, chemical, or neurogenic. In spite of their diverse aspects, they are presented together in this chapter devoted to angiospasm.

**Classification.**—Angiospastic diseases and syndromes are classified as follows:

1. Raynaud's Disease, primary
2. Raynaud's Phenomenon secondary to other diseases
3. Scleroderma
4. Frostbite, Trench Foot, Immersion Foot
5. Pernio-Chilblains, Cold Allergy
6. Traumatic Segmentary Arteriospasm
7. Vibratory Pressure Disease (Lesions due to vibration or air pressure)
8. Glomus Tumor, Angioneuromyoma, Angiosarcoma
9. Causalgia
10. Hyperhidrosis
11. Sudeck's Atrophy
12. Arteriospasm Secondary to Venous Lesions
13. Spastic Vascular Lesions due to exposure to or injection of drugs
14. Acrocyanosis
15. Cutis Marmorata or Livedo Reticularis

The spasm is activated by exposure to a tactile, thermal, emotional, environmental, or chemical irritation. There is a neurogenic and circulating chemical theory as to the method by which the reflex spasm is produced. Some believe that the entire complex can best be explained by a nervous pathway. Others contend that as the result of the stimulus some chemical or organic substance is liberated into the blood stream and transported to the area where the spasm is produced. Neither method has been proven to be the correct one to the exclusion of the other. Most investigators accept the chemical pathway as the more logical one. It is believed that adrenalin, sympathin, or some unknown substance which we call *spastin*, is liberated into the blood circulation, and due to this stimulus the part affected then goes into spasm.

Many of the arteriospastic conditions do not have an organic basis. True Raynaud's disease is in this category. Raynaud's spastic phenomena may initiate or complicate an obliterative arterial disease and its development at some time in the course of the pathologic pattern of this disease may be decisive in its outcome if not relieved. This refers particularly to occlusive arterial diseases such as arterio- and atherosclerosis. Another example is the spasm following an arterial embolism.

Other spastic phenomena may be due to the exposure to cold, pneumatic air pressure or trauma. The causes for the development of others is obscure. Unrelieved spasm will cause occlusion and develop into an organic lesion at a later date.

## PRIMARY RAYNAUD'S DISEASE AND SECONDARY RAYNAUD'S PHENOMENON OR SYNDROME

Raynaud's disease is due to a spasm of the peripheral arteries, occurring particularly in the arterioles with local arterial failure and vasomotor recurrent attacks. It may be excited by cold or some nervous reaction and involves most often the upper extremities. The spasm follows each exposure to nervous, mental, emotional, chemical or thermal stimuli to which the patient is susceptible.

The condition may be primary or it may arise secondary to an occlusive arterial disease, such as arteriosclerosis, or after frostbite.

**Etiology.**—The initial reference to spasm was by Quesnay in 1793.[13] He described it from a blood slide in 1817. Virchow gave the first real outline of the action of the reflex.

Despite the fact that Raynaud's original description was made ninety years ago (1862),[14] the diagnosis was made so rarely that only 30 true cases could be collected from the 500 reported in American and British literature up to 1936.[6] Lewis,[7] Morton and Scott,[9] Pearse,[10] Spurling,[17] Villaret,[18] Peet,[11] Allen and Brown,[2] Ray[14] and others have contributed to the literature since that time.

To simplify the diagnosis of primary Raynaud's disease, only the small group of patients which fulfill the fundamental[2,20] requirements are so classed. These are:

1. Intermittent spastic attacks with color changes which precede the trophic changes by months or years.
2. No evidence of occlusive disease to which the spasm could be secondary.
3. Trophic changes or gangrene limited in a large degree to the skin.
4. Symmetrical or bilateral involvement.
5. Disease present for a minimal period of two years.

It is obvious that all of these requirements may not be present in every patient who has Raynaud's disease but the majority of them will be evident.

The term *Raynaud's Syndrome* is reserved for patients who have an organic basis for the temporary or permanent resultant spasm. This includes that seen in thromboangiitis obliterans and arteriosclerosis obliterans as well as those exposed to cold and other environmental changes or trauma.

**Sex and Age Incidence.**—*Primary Raynaud's Disease*—Women are more subject to Raynaud's disease than men (70 per cent) Raynaud's disease appears most frequently in individuals between the ages of eighteen to thirty-five years. The patients are often of the nervous and anemic type In *secondary Raynaud's syndrome,* the sex and age follows that of the underlying organic lesion

**Symptoms.**—In *primary Raynaud's disease,* with exposure to cold, emotional stress, or other stimuli to which the patient is susceptible, the fingers blanch and turn a gray or waxy color A second phase of rubor follows These changes may occur many times a day or in some only on rare occasions



FIG 71 —Typical Raynaud's Disease Bilateral symmetrical involvement

After this condition has been present for some time, the arterial failure causes trophic changes These trophic changes are most often in the form of ulcers, which are extremely painful, the digits becoming shortened and sclerotic due to infection and tissue loss

If the disease continues, there may be necrosis and gangrene of the part The hands become cyanotic, and there may be ulcers of the finger tips These lesions are usually symmetrical In most of these patients, exposure to cold will inaugurate the spasm Atrophic changes follow

In *secondary Raynaud's syndrome,* the lesions are similar and are in addition to the lesions caused by the underlying primary disease, *i e*, arteriosclerosis or frostbite

**Pathology.**—The early pathology is unknown as pathologic observations are based on patients seen many years after the onset of the disease

Intimal thickening of the vessel occurs in all patients.[20] Usually the vessel dilates but at other times the vessels may be tightly constricted.

Whether these distal arteries fill as well as normal ones is still open to question. Allen's arteriograms indicate that this may be true.[1]

Capillary microscopy in many instances is diagnostic, the capillary loops being large and the blood cells passing through these loops slowly and irregularly.

Where necrosis has occurred the typical pathologic picture of ulceration and tissue destruction is seen.



Fig. 72.—Normal capillaries. Seen through capillary microscope. (Courtesy Dr. A. Wilbur Duryee.)



Fig. 73.—Capillaries in Raynaud's Disease. Widely dilated capillaries with slowly moving irregular-sized red cells passing through them. (Courtesy of Dr. A. Wilbur Duryee.)

The pathology of secondary Raynaud's syndrome is that of the underlying primary disease, with the additional changes due to spasm The additional pathology depends on the length of time spasm has been present

DIAGNOSIS —A *Primary* —In primary Raynaud's disease, occlusive arterial disease must be ruled out The sex incidence (70 per cent of females), the age of the patient (usually young), bilateral involvement of the upper extremities and symmetry of the lesions, and the response to the exciting stimuli are all diagnostic points The hyper-reaction of allergic, sensitive and neurotic individuals to slightly abnormal stimuli must be ruled out Unusual reactions due to bashfulness, diffidence or excessively modest reactions, at times, are borderline to spasm

Capillary microscopy should be performed by a trained individual, and if correlated with clinical findings, will complete the diagnosis The response to sympathetic blocks, clinically, and by skin temperature readings, may help the diagnosis

TABLE 21 —DIFFERENTIAL DIAGNOSIS OF VASCULAR DISEASES OF THE EXTREMITIES

| | *Thromboangiitis Obliterans* | *Arteriosclerosis* | *Raynaud's Disease* | *Erythromelalgia* |
|---|---|---|---|---|
| Sex | Males 99% | Males 90% | Females 85% | Females 70% |
| Age | 25–45 years | 45–85 years | 17–35 years | 30–50 years |
| Race | Hebrews predominate | Any | Any | Any |
| Claudication | +++ | Usual | Absent | Absent |
| Rest Pain | Severe +++ | Mild | Absent | Variable |
| Type of Rest Pain | Sharp, stinging | Aching | Absent | Burning |
| Rubor on Dependency | +++ | +++ | Absent | Absent |
| Pallor on Elevation | +++ | +++ | Absent | Absent |
| Tobacco | ++++ | +++ | Frequent | ? ? ? ? |

B. *Secondary Raynaud's Syndrome* —The underlying organic disease must be diagnosed by its symptoms Many of the cases of secondary Raynaud's syndrome are seen in the lower extremity

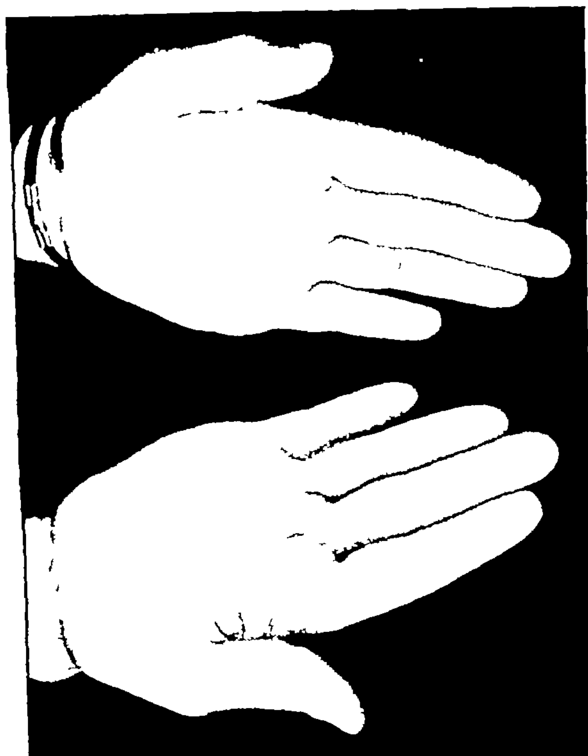
The differential diagnosis of vascular and vasospastic diseases is summarized in Tables 21 and 22

**Treatment.**—In primary Raynaud's disease, the treatment varies with the degree of symptoms, the severity of the disease, and the stage at which the individual is seen If the symptoms are mild, no therapy may be required In advanced lesions sympathectomy is necessary.

Secondary Raynaud's spasm requires therapy for the primary lesion and often sympathectomy.

1 **Prophylactic Measures.**—In primary Raynaud's disease, the protection of the individual against undue exposure to cold, fatigue, and nervous excitement is important not only prophylactically but also as part of the conservative therapy regimen The elimination of exciting irritants such as nervous upsets, emotional crises, and the avoidance of severe temperature changes may be all that is necessary to control the mild cases If the patient's occupation subjects him to such stimuli, or if there is direct trauma to the involved part or exposure to cold, the type of work must be changed

## PLATE I



Raynaud's Disease. Stage of rubor (Courtesy of Dr Irving S Wright from Stroud's, "Diagnosis and Treatment of Cardiovascular Disease," F A Davis Co )

PLATE II



Raynaud's Disease stage of spasm. (Courtesy of Dr Irving S. Wright from Stroud's, "Diagnosis and Treatment of Cardiovascular Disease," F. A. Davis Co.)

TABLE 22.—DIFFERENTIAL DIAGNOSIS OF OCCLUSIVE VASCULAR AND VASOSPASTIC DISEASES

| *Disease* | *Age* | *Sex* | *Part of Body* | *Early Signs* | *Late Signs* | *Arteries* | *Veins* |
|---|---|---|---|---|---|---|---|
| Raynaud's Disease | 15–45 (65%—12-35 years) | F | Upper | Color change | Necrosis | Large arteries are normal. Capillaries involved | Normal |
| Pernio | 15–45 | F | Legs | Burning, itching | Necrosis | Normal | Normal |
| Scleroderma | 10–50 | F | Upper and lower face etc | Shiny skin spasm | Necrosis hardness retraction | Obliteration | Secondary obliteration |
| Acrocyanosis | 12–30 | F | Upper | Cyanosis on dependency | Marked cyanosis Infection | Normal | Secondary obliteration |
| Thromboangiitis obliterans | 18–45 | M (Almost unknown in females) | Lower Extremities | Rubor pallor claudication paresthesia | Necrosis or gangrene | Obliteration | Phlebitis in one-third |
| Arteriosclerosis | 35 + (40% of males over 40, have arteriosclerosis) | M | Lower Extremities | Early ulcers same as thromboangiitis obliterans | Infection and gangrene | Obliteration calcified | Normal |

Other therapy will fail if this change is not effected. In secondary Raynaud's syndrome, prophylactic therapy may be decisive.

2. **Medical Treatment.**—In primary Raynaud's disease, mild sedation and the vasodilating drugs of questionable value elsewhere may be sufficient to relieve the individual who suffers only slight spasm.

The cause of an anemia, if present, should be determined and eliminated. Those with psychiatric problems should have competent psychiatric care. The local vasodilation afforded by "Mecholyl" given by iontophoresis may be helpful. Certain so-called vasodilating drugs, such as the nitrites and papaverine, in large doses may be beneficial. Sometimes a change of climate helps. Whirlpool baths, light massage, and corrective, but mild, exercises may bring relief. Nonconstricting gloves or resilient pads may solve the problem of occupational exposure. Smoking should be discontinued permanently by all such patients.

Vitamins should be given to those who appear deficient. If there are hypoglandular or menopausal symptoms present or these states are suspected, these should be corrected by the administration of the necessary hormones. Efforts to ameliorate this disease with ACTH or cortisone have not been effective in the few patients reported. At times, a remission occurs but the general course of the disease continues.[5,12]

A few patients will respond to such drugs as tetraethylammonium chloride, "Priscoline," and perhaps "Dibenamine," but the results vary and are not consistent. The final place of such drugs in the therapy of vasospastic diseases is still questionable. The use of this type of drug is indicated as an aid, especially in upper arm lesions where sympathectomy is not as satisfactory.

*Other Drugs.*—Oranixon (3-o-toloxy-1, 2-propanediol) *—This drug of anesthetic derivation may act in one of two ways. It may cause muscle relaxation, locally in the muscle or in the smooth muscle of the blood vessel since it has been effective in the spasm and convulsions of tetanus. A possible second mode of action may be that of sympathetic paralysis similar to that of other anesthetic agents. It has been tested in our Vascular Clinic, clinically, both in organic and spastic arterial diseases with confirmed findings of improvement. While such results must be based on patients' and observers' interpretations, a definite pattern of improvement was obtained in over 70 per cent of the patients. The use of placebos was correctly discovered by this percentage of the patients. Correlation of the improvement by skin temperature, oscillometric and claudiometer readings was inconclusive. Similar efforts to coordinate clinical findings and such tests with other therapeutic modalities usually have failed.

Roniacol Tartrate.—Tartaric acid ester of beta-pyridyl-carbinol (the alcohol corresponding to nicotinic acid). This drug produces a flushing and feeling of warmth. Whether it actually dilates vessels more than temporarily is open to question. Its side effects are minimal and it may be of minor beneficial effect. Possibly, its entire effect can be attributed to its alcoholic content.

Nicotinic Acid.—Nicotinic acid is one of the vitamin B complexes, also called niacin. Its formula is $C_5H_4N-(COOH)$. This drug has a questionable vasodilation effect.

* Organon Inc., Orange, New Jersey.

Others.—Intravenous ether procaine and other anesthetic substances have a temporary dilatation effect and may be indicated diagnostically or, for a short time therapeutically

*Sympathetic Blocks*—The ganglia involved i e the stellate and the upper thoracic for the hands and arms may be blocked for a short time by the injection of an anesthetic solution This may be indicated therapeutically in an acute phase or diagnostically Prognostically it may indicate the effect to be expected from surgical sympathectomy

3 **Surgical Treatment**—A *Primary Raynaud's Disease*—If the symptoms progress despite conservative measures an operation should be considered (25 per cent)

The results from surgical sympathectomy in the upper extremities are not as consistent as they are in the lower extremities

*Sympathectomy for Raynaud's Disease*—The operation of preganglionic sympathectomy as advocated by Smithwick has been used through the years since it was suggested in 1936[14] Denervation and not excision of the ganglia was advised upon theoretical grounds In many instances, however the ganglia would be excised inadvertently Some of these patients seemed to obtain better results than those in which the ganglia were preserved The sensitization to adrenalin which was supposed to follow dorsal ganglionectomy has not been observed with consistency In some patients therefore excision of the ganglia in addition to the preganglionic denervation has been the treatment of choice In each instance a section of the second and third dorsal intercostal nerves has been removed The results compare favorably with those reported by Blaine Coller and Carver[2] The 64 per cent recurrence reported by Felder[1] and his co-workers after a typical so-called preganglionectomy certainly must be improved Complete denervation is the important part of the therapy in the advanced cases and in our hands has been best accomplished by wide resections of both pre- and post-ganglionic types Again it is emphasized that surgical treatment is required in only those patients who develop gangrene have tissue loss or have progression of symptoms despite conservative therapy It is therefore performed in only 1 out of 3 or 4 patients For details on this technic see the chapter on Interruption of the Sympathetics (p 487) The progress of the disease to irreversible necrotic and organic changes should hasten surgical intervention before this occurs.

Other surgical therapy includes surgical dressing of ulcers drainage of purulent collections and occasionally amputation of a necrotic fingertip Conservatism should be the surgical rule in the treatment of an ulcer or a necrotic area. Soaks antibiotic therapy daily dressings and painless removal of small areas of sloughs from day to day permit most of these wounds to heal by epithelialization

B *Secondary Raynaud's Syndrome*—The surgical therapy of Raynaud's syndrome complicating an organic lesion such as occlusive arterial disease includes treatment of the basic lesion but care of this spastic part may markedly alter the course of the underlying disease This subject has been discussed in the chapter on Occlusive Arterial Diseases (p 190)

Where the spasm is a factor or where the response to sympathetic blocks indicates that the patient will be improved by such measures an

early sympathectomy should be performed In many patients, even where spasm cannot be proven to be playing a part, sympathectomy has resolved the process It is apparent, therefore, that mere dilatation of the capillary bed, even in the absence of clinically demonstrable spasm, is of therapeutic value For details, see the chapter on Interruption of the Sympathetics (p 487)

## SCLERODERMA (ACROSCLEROSIS OR ACROSCLERODERMA)

**Etiology.**—The cause of scleroderma is unknown This condition is frequently initiated with Raynaud's syndrome, because often the two conditions simultaneously appear Some vasospasm occurs in all patients who have scleroderma There are two forms of the disease A local type is characterized by single or multiple areas usually restricted to the extremities There are frequent remissions and it is in this group that cures have been reported The other form is more *diffuse* It attacks many parts of the body and has visceral manifestations Its effect on the kidney, lower esophagus or heart may be fulminating and suddenly fatal The face, neck and chest are frequent areas affected, in addition to the extremities In its final stages, the skin becomes hard and tense and thickened, with marked fibrosis Atrophic changes occur Scleroderma may involve any part of the body When it is seen on the face, it causes a characteristic fixed facies in which the hard shininess of the thickened skin may be associated with retracted eyelids and contracted nares and oral apertures

Scleroderma was considered a disease of the skin alone, being first described in the seventeenth century by Curzio That its underlying basis is arterial disease is a more recent concept The theories as to the origin of scleroderma are multiple and have been summarized by Duryee[25] as: (1) endocrine, (2) nervous, (3) toxic or infectious, (4) traumatic, and (5) vascular Any or all of these may be the originating factors, or play a part

The generalized reaction and the involvement of nearly all parts of the body at different stages support a vascular etiology[24-27] as the underlying cause

Allen[21] suggests that scleroderma is related to some abnormal formation or metabolism of the collagenous tissue, particularly in the cutaneous and subcutaneous layers If it is a disease of the collagens, it does not respond like others in this group to cortisone or ACTH

Theoretical conjectures that the disease is due to a parathyroid glandular imbalance have not been proven

*Sex and Age Incidence*—Women and children are most often affected

**Symptoms.**—The early symptoms of scleroderma may be those of a mild localized Raynaud's type of spasm The skin soon becomes shiny, somewhat brawny and indurated. It may be intensely pigmented in part, but at times it loses its pigment and becomes white like vitiligo There may be a mixture of over and under normal pigmentation

As the disease progresses, the skin's hardness increases and contractures occur as the underlying fascia is involved Since these changes occur most often near joints, flexion contractures are common deformities The skin

on the extensor side of the joints becomes thin avascular, and frequently breaks open and is the site of repeated infections and ulcerations Mild trauma causes skin breaks Ulcers recur and cellulitis spreads from these secondary infections due to the limited defensive ability of the part Tendons and even joints may be exposed

The contraction on the face may pull the eyelids nares or mouth down, the eye areas producing ectropion The skin becomes board like thin and transparent As mentioned the other organs of the body may become affected These include the heart kidneys the lungs the intestinal tract and particularly the esophagus

**Treatment.**—1 Prophylactic Measures.—If exposure to chill or cold is suspected as being the cause this must be avoided If the patient's occupation exposes him to an irritating agent to which he is sensitive the

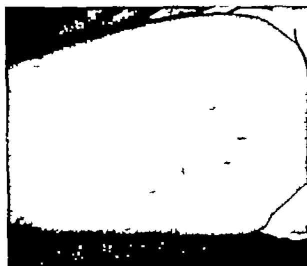


Fig 74 —Localized scleroderma on the thigh. Induration and retraction of the is typical. This patient was treated by local excision and sliding skin graft, wit recurrence in twelve years.

work must be changed In mild cases a change of climate appears to h effected some cures Smoking should be discontinued permanently ' diet of the patient should be high in vitamins and minerals

All preventive measures must be utilized Drugs endocrines vitam and surgery do not affect the course of the disease in most patients.

Irritating stimuli in the patient s environment reducing the incid of infections and preventing and correcting disability from contracti often is all that can be done

(a) *Mecholyl' by Iontophoresis* — Mecholyl (acetyl beta metl choline chloride) by iontophoresis will cause some degree of improvem in about 60 per cent of the patients [14,23] Our experiences were not so sa factory only 20 per cent were helped The action of this drug appare increases the blood supply in the diseased part

A general reaction to Mecholyl also occurs. There is an increas pulse dizziness and light headedness, blushing flushing of the skin faintness

(*b*) *Other Drugs* —Papaverine hydrochloride in large doses combined with a sedative is beneficial to some patients. More recently, para-aminobenzoic acid ($NH_2C_6H_4$-COOH), a member of the vitamin B group, has been advocated as a therapeutic measure following its use in lupus erythematosus At times, this has been combined with one of the sulfa drugs The dosage has varied, but in general 6 to 8 grams of the drug in the form of sodium or potassium para-aminobenzoic acid have been given Encouraging results were reported [22,42] We were unable to demonstrate definite or permanent improvement in any patient Since ill effects are rare, its trial is indicated The combination does seem to have beneficial effects in other collagen disorders and it has not been proven that there is only one form of scleroderma

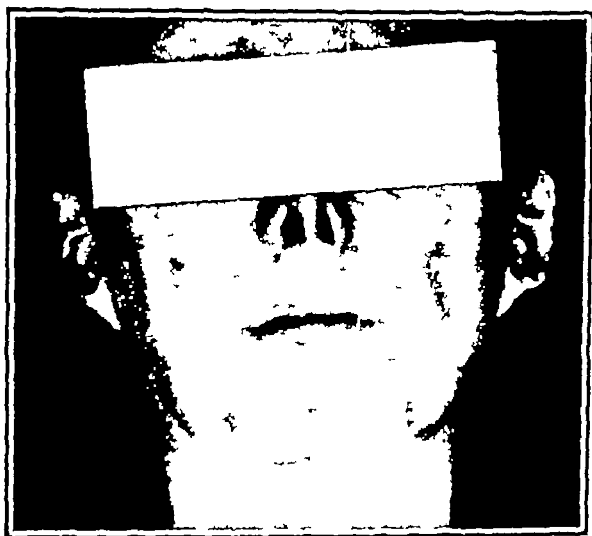


FIG 75 —Shows typical scleroderma changes in the face Note ectropian changes in the nares and the retraction of skin of face and ears

(*c*) *Possible Other Therapy* —1 Sympatholytic and Adrenolytic Drugs —Relief in these diseases by such substances as the adrenolytic or sympatholytic drugs has been tried, but, in general, they have not been effective Priscoline, tetraethylammonium chloride and dibenamine theoretically should help, but practically, the results are discouraging. Further trial of these drugs is advisable

2 Adrenal Cortex —Adrenal cortex replacements such as cortisone, or adrenal cortex stimulants such as ACTH, have been given extensive trials No consistent or persistent benefit has been demonstrated, and in some cases, more rapid progression of the disease has been reported

(*d*) *Endocrine Therapy* —Improvement in patients with scleroderma after the administration of estrogens has been reported as due to their effect on calcium metabolism [24]

It has been observed that testosterone propionate increases the normal vascularity of the skin in some patients deficient in this hormone [??]

Thyroid extract[18] has been given to patients with scleroderma by many investigators on the basis that it improves the dry coarse skin and increases the capillary blood flow in thyroid-deficient individuals Thyroid extract may be used empirically, but the results have not been good

In treating patients with scleroderma it is suggested that estrogens testosterone and thyroid be given to any patient who appears deficient in hormones

(e) *Vitamin Therapy*.—Patients with scleroderma have been found to be deficient particularly in vitamin C.[23] This may be due to the local





Fig 76 Fig. 77

Fig 76.—Advanced sclerodermic changes in a child of fourteen. Nearly every part of the body was involved and the condition progressed despite all types of therapy. Sclerodermic vascular changes of the kidneys caused marked hypertension with uremia and death.

Fig 77.—Child age three with the skin changes typical of scleroderma. Scleroderma may have been secondary to the occlusion of the artery of an arteriovenous aneurysm.

changes interfering with vitamin C absorption. The use of large doses of vitamin C is indicated in this condition.

Massive doses of vitamin B (Thiamine hydrochloride) and especially $B_1$ and $B_{12}$ also have been given empirically because of the primary or secondary involvement of the central nervous system. The pellagra like pigmentation in scleroderma also would argue for some vitamin B

complex deficiency, and would warrant the administration of such vitamin supplement in therapeutic dosage Vitamin E has been tried in this condition as in nearly all others

2 SURGICAL TREATMENT —If the symptoms are severe and progressive despite medical measures, some type of operation may be considered.

(a) *Sympathectomy* —In early lesions, especially if Raynaud's syndrome plays a prominent part in the symptoms, sympathectomy is indicated

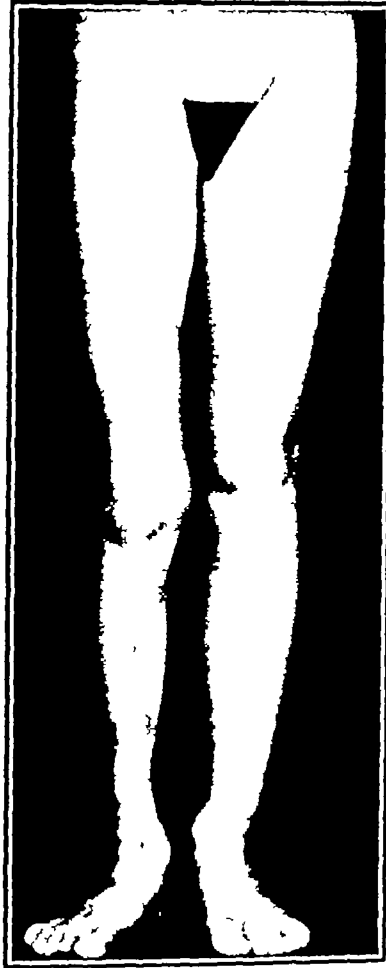


FIG 78 —Same child as in Figure 77, four years later Note scleroderma changes confined to extremity and groin Sympathectomy and physiotherapy released many contractions Note shortening of right leg and tilt of pelvis

Sympathectomy cannot be considered curative, but by increasing the capillary blood supply, some of the local skin changes and infections may be controlled Where the skin lesions are not advanced, spasm can be relieved It must be emphasized that the sympathectomy is only an additional therapeutic aid and it probably will not alter startlingly the progress of the disease

In 1939, Bernheim and Garlock,[23] and again in 1941, Johnson[24] reported that the vasodilation following sympathectomy in scleroderma was only of temporary duration Like vasodilation after sympathectomy in any disease, we believe if the results are short-lived it indicates that an inadequate

operation has been performed Several of our patients have had their disease remitted for many years after a complete sympathetic denervation Progression of the disease does not mean that the sympathectomy effect was short lived In advanced cases no benefit can be expected from interrupting the sympathetics While sympathectomy does not halt the process, the locally increased vascularity may ease the symptoms

(b) *Parathyroidectomy*[23, 34, 40]—Of the three parathyroidectomies discussed by Duryee in a series of 120 patients with scleroderma, 2 failed to alter the course of the disease In 1 patient in whom the operation was so complete



Fig 79 —Roentgen ray shows the sclerodermic changes at the lower end of the esophagus typical of scleroderma. Spasm and stricture may occur

that tetany developed it was thought that some temporary improvement resulted We were unable to substantiate in our experience the good results reported by Oppel[34] and other investigators Unless there are definite signs of hyperparathyroidism this operation is not indicated

(c) *Excision* —In some patients with scleroderma a single local lesion will appear If the lesion is localized early excision may be sufficient to cure it. In one such patient seen early after the onset of the disease the lesion was limited to a 3 × 6 inch area near the hip This lesion at the hip was widely excised and the defect repaired by a sliding graft. There has been no recurrence at the excised area in twelve years (See Fig 74 )

It is recommended that if an early lesion is observed at a site where it can be surgically removed, excision should be considered by the surgeon

(d) *Excision and Skin Graft* —A possibility of eradication of the disease by radical excision similar to that employed in cancer is feasible when the disease is localized The electric dermatome, which has made skin grafting simple, makes this surgical approach easier The diseased area can be excised to include the fascia in which much of the contracture occurs and fresh, nondiseased skin can be placed on a denuded recipient muscle base

Other types of operations have been unsuccessful

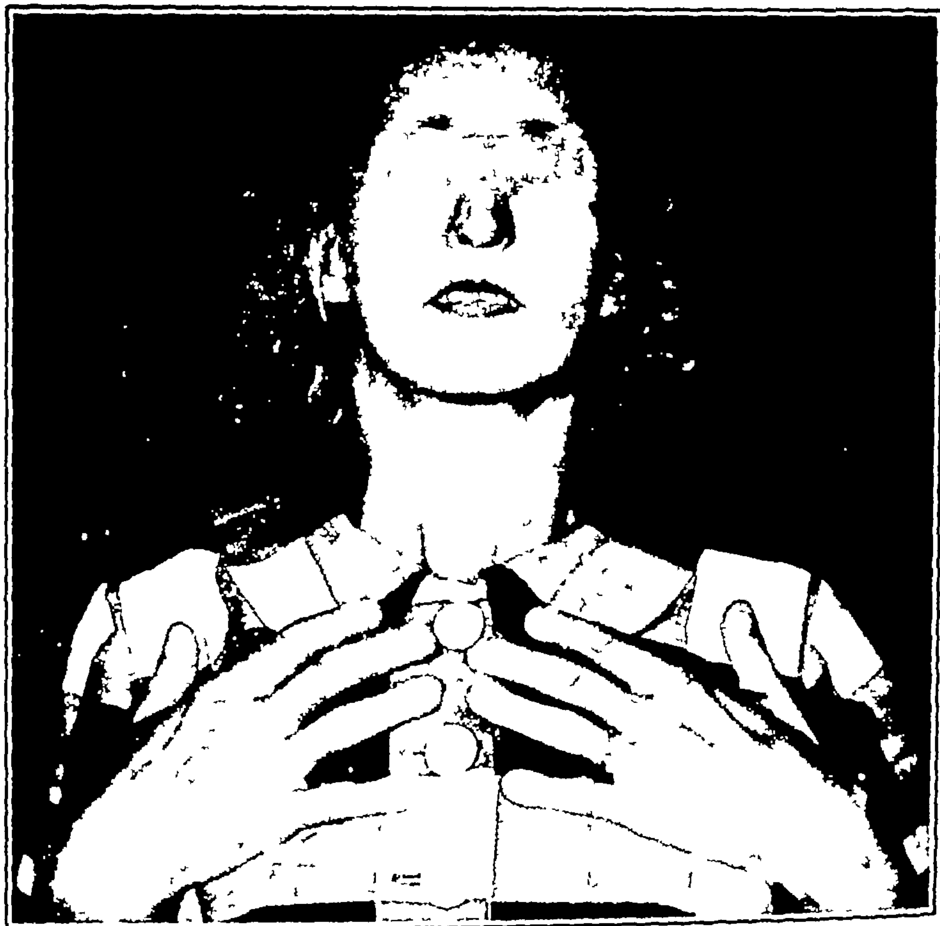


FIG. 80 —Combined scleroderma and Raynaud's disease Findings typical of both lesions Treatment Surgical sympathectomy with partial relief of symptoms

*Physiotherapy with graded exercises*, active and passive motion and whirlpool may keep the part functioning for years Braces, extension apparatus and *manipulation* as the joints stiffen may be required *Surgical care of repeated infections* usually is necessary, and this should be ultra-conservative

In summary, surgical therapy of scleroderma is unsatisfactory and inadequate to prevent, or sometimes even to delay, the inevitable course of the disease

## FROSTBITE, TRENCH FOOT, IMMERSION FOOT

Our concepts of the effect of cold on extremities were greatly changed as the result of our experience in the recent war, and the exposures in the

Korean campaign again have altered our therapeutic program In the early days of World War II when many ships sank in the northern Altantic as a result of submarine warfare a large number of men were exposed to varying degrees of temperature for considerable time These thermal changes were combined with trauma, submersion dependency of the part starvation, and strain The importance of the treatment of these patients was realized early in the war, and through the cooperation of the British and American medical services these patients were segregated at once in special hospitals on both sides of the Atlantic for observation and treatment On this experimental evidence and the Korean campaign much of our modern therapy of frostbite trench foot and immersion foot is based While the last word in therapy of these lesions is far from written a more rational basis for treatment is now available

**Frostbite** —ETIOLOGY —The effects of exposure to cold depend upon the degree of cold the length of time of exposure the presence or absence of trauma or immersion the age, weight and physical status of the one exposed and the type of immediate and late therapy When the body is exposed to cold the central portion rarely cools below 37° C (rectal temperature) The peripheral and superficial part of the body however may fall below this critical temperature The presence or lack of wind the humidity and the patient s clothing as well as his general condition and age are important. If the part is wet, the danger of freezing increases Dependency wet and hypoproteinemia as well as exhaustion trauma and inactivity all play a part.

Exposure to a more moderate degree of cold over a longer period of time can cause frostbite It can occur at 8° F or at temperatures from 8 to 14 F in the presence of a strong wind The incidence of frostbite at a temperature of 8° F is 5 in 10 000 while at 8° to 24° F frostbite occurs only if there is a strong wind [46]

The exact temperature at which freezing occurs varies with the individual There are innumerable individual variations between 4° C (34 8° F) and −10 C (14° F) In some persons a temperature as low as −20 C (−4° F) has not caused freezing

Above 24° F frostbite is rare unless the patient has an underlying defective arterial system A sudden change of temperature such as occurs on entering refrigerators or refrigerator cars is also an etiologic factor

The exigencies of war create a special frostbite problem This is true particularly in defensive fighting or during a retreat. In either category the individual s physical status hygiene and personal care deteriorate and discipline which is a primary requirement prophylactically becomes lax. Examples are Napoleon s retreat from Moscow the Battle of the Bulge in World War II and the recent retreat of the American forces and particularly the Marines after they were cut off at the Changjin Reservoir in Korea [36] Reports from Korea sickening to most Americans pointed up the problem of frostbite and made dissemination of knowledge on how to treat it of timely importance Frostbite always has been of military and naval importance particularly since the days of the Romans and reached extreme prominence in the failure of Napoleon s army to survive its retreat from Moscow To the extent of the loss of

heart of his trained personnel, cold played its part in his subsequent Waterloo In World War I, the treatment of the condition was recognized as paramount when the soldiers had to stand in trenches for an indefinite time under wet and cold conditions In World War II, the problem was extremely important in the Aleutian Islands campaign, and in the Battle of the Bulge The experiences of both the British and American naval forces with immersion foot, which is the naval counterpart of trench foot, became significant This followed the large number of ships sunk on the North Atlantic and Murmansk runs in the early days of the war, and the subsequent exposure of the survivors in open lifeboats and rafts at sea The effect of cold on the Germans in World War II is shown by the following In one eight-month period in 1941–1942 in the Eastern Zone there were 200,000 dead, 700,000 wounded and 45,000 missing, of which total 30,000 were officers In the same period 112,000 were incapacitated by freezing or frostbite (12 divisions) [Joseph Goebbels, in his captured diary] It is apparent that this problem has been a factor in the fate of wars and of nations for an indefinite period of time, and again it may become decisive, as we may face another campaign which likely will be fought in the Arctic or near-Arctic zones

The problem of frostbite in civilian life exists and increases with longevity. Those working out of doors, under freezing conditions, and in refrigerator cars or air conditioned meat plants, are exposed Now that many older-aged people continue to work, the incidence of civilian frostbite is increased due to arteriosclerotic changes in their vessels When subjected to cold, even above freezing temperatures, they may develop lesions similar to those experienced by younger individuals under freezing conditions In the presence of moisture, or a cold wind, it is not necessary for the temperature to reach actual freezing levels to produce the frostbite lesions In addition, if movement of the parts is restricted, as when military personnel are "pinned down" by enemy fire or inactivated for other strategic reasons, the lesion will develop more rapidly Poor hygiene, the inability to change to dry and clean clothing, and the trauma of walking after the lesion is present, all are contributing and complicating factors The part that hygiene plays was illustrated well by the casualties after the invasion of the Aleutian Island of Attu In that campaign, the opposing sides were in freezing and wet conditions for ten to fifteen days without the opportunity to change to dry and clean stockings and shoes This condition resulted in a fairly high amputation rate (24 per cent in the Americans) while in the captured Japanese, the amputation rate was close to 100 per cent This figure denotes the factor of better equipment, better hygiene, and better care as of additional importance in the prognosis. The difference between trench foot and immersion foot and frostbite is only of etiologic importance When the limb is cold and/or wet and the part is subjected to some trauma, such as walking, one terms it "trench foot" When the foot is cool or wet, without trauma, and dependent such as occurs when sitting in a lifeboat for an indefinite period of time, the lesion is called "immersion foot" In the dependent position, as when in the lifeboats, certain other factors such as edema, starvation, hypoproteinemia, and vitamin deficiency all play a part The amputation rate however in

# PLATE III





Severe frostbite both feet Superficial tissue separating Treatment surgically conservative (Courtesy Surgical Service U S N *Hospital, Philadelphia, Pa* )

Superficial gangrene of hand Conservative therapy with recovery of all but end digits. (Courtesy Surgical Service U S N Hospital, Philadelphia, Pa )

Originally published in Gen Practitioner 1953

immersion foot is relatively low Without any such military surroundings the end result is called frostbite

SYMPTOMS —The symptoms of frostbite vary with the above stated factors. A poor physical status, such as anemia dehydration and malnutrition as well as smoking thereafter contributes to the severity of the condition The degree to which physical status contributes to the incidence was well shown by the experiments in which nearly all of the rats under 19 grams in weight died before their tails were frozen while the majority of those weighing over 25 grams survived the cooling from −90° to −10° C[44]

There may be no symptoms. Fifty per cent of the Marine casualties were unaware of their lesions

Erythema follows mild exposure The early symptoms of frostbite are those of spasm with the part becoming white pale and cold Following this initial change there may be some bluish discoloration as venous congestion develops Thereafter there is some edema due to this venous congestion. The part becomes cold somewhat discolored and gradually a mottling may occur If the cold persists the part may become pale and marble white

Frostbite may be classified clinically like burns dependent upon the severity of the tissue changes[45] This has been modified

1 *First Degree Frostbite* —In first degree frostbite the part is subjected to intense cold for a short time or to a lesser degree of cold for a longer time *The patient recovers completely although there may be some* residual hypersensitivity to cold exposure for a considerable time thereafter Symptoms of first degree frostbite are erythema followed by spasm with a burning and tingling sensation or anesthesia in the parts.

2 *Second Degree Frostbite* —In second degree frostbite the patient is exposed to cold for a longer period of time or to a more extreme degree of cold than the patient with first degree frostbite Recovery is accompanied by loss of some tissues particularly the skin and at times some of the subcutaneous structures. The symptoms are caused by ice crystals in the *tissues Anesthesia and paresthesia occur As the part warms there is a* marked reactive hyperemia Wheals and blebs develop and there are secondary sloughs There may be secondary infections as the part recovers Pressure or trauma aggravates the symptoms and increases the tissue loss

3 *Third Degree Frostbite* —In third degree frostbite the part is subjected to extreme cold for a long period of time Such cases do not recover completely The skin and subcutaneous structures are lost and there may be a loss of tendons bones or even of the entire extremity If a large part of the body is so exposed death will result Trauma plays an important part in the degree of destruction. Immobilization increases the signs For example in the Marines who suffered frostbite in the retreat from the Changjin Reservoir in Korea gangrene of the heels was most common in those who rode out These individuals wiggled' their toes but rested on their heels thus adding the trauma of constant pressure with its avascularity to the cold The part that briefing and discipline may play prophylactically was well shown by the reports of these survivors The

troops were young and inexperienced in cold exposures. One after another told of striking his feet with his gun butt as numbness developed in an effort to restore the "feeling." Each such trauma increased the necrosis.[46]

In third degree frostbite, the vascularity to the part is permanently interrupted, and thrombosis occurs in the blood vessels. As the part is warmed, necrosis and gangrene often develop. Such destruction, despite experimental studies, has occurred regardless of the length of time and precautions taken to bring the temperature back to normal.

4. *Exposure to Cold With Anoxemia Due to High Altitude Flying.*—During modern war, where high altitude flying is necessary, the exposure to cold may be extreme, the temperatures ranging from −40° to −60° F.

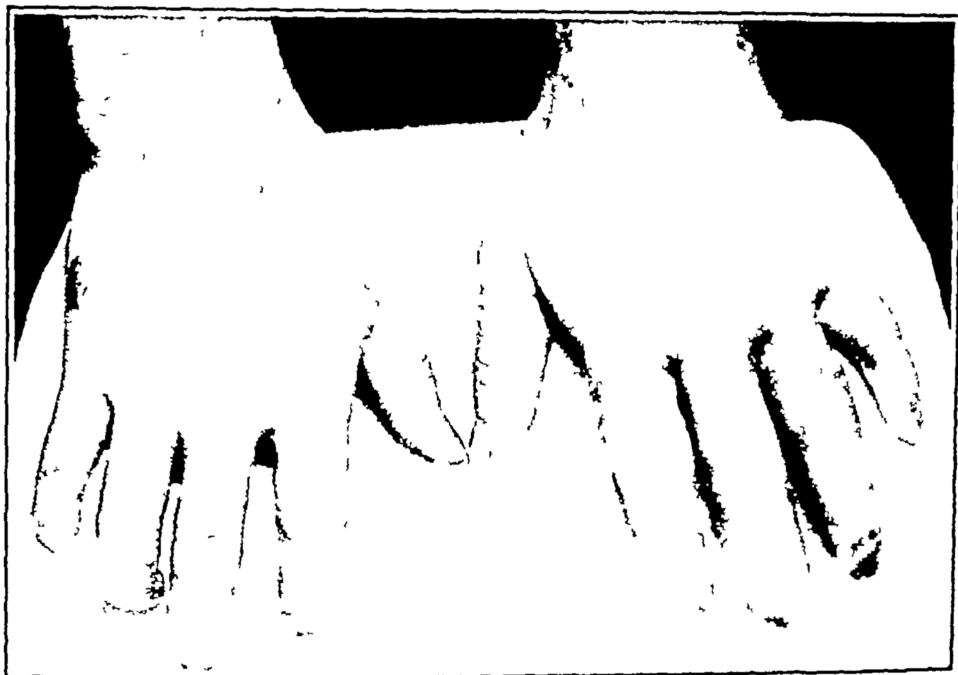


Fig. 81.—Cold allergy. The hand on the right was immersed in ice water for one minute. This was followed by swelling, edema, redness and blanching.

When the air-tight control is broken by gunfire or accident, the crew is subject to sudden and extreme changes in temperature. Associated with such falls in temperature is a marked anoxemia, to which many of the subsequent symptoms are due. This exposure is like the deep freezing of foods.[47] Similar exposures may occur in commercial or private flying. High flying causes frostbite aided by a lack of oxygen. When anoxemia and cold are combined, the destruction of tissue may be more rapid. In this type of exposure, the symptoms vary from mild erythema to blanching and whiteness and even vesicle formation, in extreme cases, rapid necrosis and gangrene develop.

Defreezing in these individuals, if sudden, is similar to deep freezing. Some cellular destruction is inevitable in such cases in the return to a normal temperature. The skin in these cases becomes shiny and tense with subcutaneous hemorrhages as the blood vessel walls are destroyed. Later gangrene develops; this is of the atrophic type with a dry, shriveled extremity.

The reason the extremities are involved while the face and other exposed parts are spared probably is due to the relative decrease in the blood supply to the arms and legs.

Prevention of such cases of frostbite is extremely important The possibility that the temperature within the plane will fall due to shell fire damage or accidental breaking of the closed compartment housing the pilot and crew must be anticipated The planes of the future must be compartmented as are ships to solve this problem The crew then could move to a different compartment should the soundness and safety of one be interrupted This would require secondary control instruments

The use of anticoagulants in these exposure patients is of importance in the recovery period The fact that an underlying arterial disease may be playing a part in producing the symptoms always should be considered

Pathology —The pathology which follows exposure to varying degrees of cold is under investigation at this time. The reports both as to the tissue changes and the reactions to various types of therapy are confusing contradictory and certainly not established The reasons for these apparently opposing reports may be due to the fact that similar conditions to those which induce frostbite in man are not similar to those used in the experimental animal

Pathologic tissue abnormalities following freezing are similar to those after exposure to heat Mild exposure causes an increased arterial flow This erythema and a damaged capillary bed produce a fluid beyond the ability of the part to remove it. This fluid accumulation is increased by a lymph stasis If the freezing is continued the tissues become fixed and when solidly frozen are hard white and opaque [49,53]

Serious pathologic changes occur when the tissues return to normal The venous and lymphatic congestion produces edema in varying degrees Large blebs follow and then gangrene develops.

Varying degrees of trauma change the reaction to the exposure The gangrene when it develops is of a cyanotic avascular type leading to a black dry gangrene Infection will alter the picture and pathology and is of enormous importance prophylactically and prognostically

The degree and rate of return of the arterial supply with warming varies dependent upon the extent and type of exposure the reaction of the individual and the rapidity of the warming In some instances arterial thrombosis is irreversible and necrosis is inevitable The return of the arterial supply when such is possible usually is more rapid than the venous and lymphatic systems can carry away The pathology as seen in experiences in the Yukon area was similar to those described Major loss of tissue or death of the part or the individual himself followed any prolonged exposure despite the method of therapy then available These therapeutic measures in general were stimulation warmth suppurative and symptomatic treatment with amputation too often and too early employed The pathology thus can vary from erythema to gangrene and from mild spasm to complete occlusion and thrombosis Maceration and destruction of the protoplasm of the blood is possible Mild to severe changes may occur in the walls and particularly the endothelium of the capillaries the arterioles and arteries the venous and lymphatic compo-

nents and secondarily the soft tissues All may be involved in varying degrees The nerves may be secondarily affected.

For years it has been taught that the tissue injury was secondary to a vascular lesion—vasoconstriction, vasodilatation, blood sludge and thrombosis. A new theory is that the injury is due to the effect of the cold directly on tissue cells This placed any vascular changes in a secondary category It is our belief that the pathology is both a tissue cell injury and ischemia caused by clotting in the peripheral vessels The erythema occurring when the part becomes red, combined with a damaged capillary bed, retards venous flow and the removal of tissue and lymph fluid. The gangrene is of the cyanotic avascular type which is "sock" - or "glove"-like and often superficial. It may be shed by the patient Infection alters the picture materially.



FIG 82 —The fingers of hands after exposure to cold in a patient who is susceptible to cold A coincidental syphilitic infection exists in this individual Previous exposure to cold had resulted in amputation of finger Others exposed to the same degree of cold were unaffected (Courtesy Dr A Wilbur Duryee)

The cold exposure problem again is pointed up by the reports from Washington—that of the first 83,000 casualties announced in Korea, nearly half were of a noncombatant nature, and nearly all of these were due to "cold casualties."

The treatment of this condition can be divided into four parts: prophylactic, the immediate treatment after the exposure, the period from the second to the twelfth week, and the therapy of the end pathology.

TREATMENT- 1 PROPHYLACTIC MEASURES - The prevention of frostbite in civilian life is important No one who is elderly or who has an occlusive or spastic arterial lesion or diabetes mellitus should be permitted to work out-of-doors in cold or wet weather The wearing of warm clothing is

essential Wind breaks for those working out-of-doors should be arranged Those who enter a refrigerator or refrigerated car for a short time may overlook the importance of this factor

When the patient has been exposed to cold over a long period of time therapy should begin at once with loosening constrictive clothing and avoiding further cold wetting of the part or trauma

Prophylactically the treatment depends on several factors

(a) *Discipline* —Discipline with strict obedience to orders and regulations about care of the feet is of utmost importance particularly in military exposure Americans are independent and careless and may refuse to obey rules which inconvenience them The British who have severe discipline and rules had a much lower incidence of frostbite in the Battle of the Bulge than the Americans 20 000 British versus 34 000 Americans Five hundred men were evacuated weekly by the American Air Force for high altitude frostbite One out of ten were pilots each of whom cost $50 000 to train Most of these casualties were due to faulty electric equipment which was caused by throwing the suits on the floor losing gloves etc In the Royal Air Force it was an automatic court martial offense to misuse such equipment and frostbite was rare unless the plane was struck by flak Lack of discipline was evidenced by men exposed to cold who told of stomping on their numb feet with their rifle butts because they could not feel them The importance of disciplinary measures applies to disasters in civilian life In individual instances the discipline must be self-imposed

Certain other points are to be emphasized

(b) *Wearing Gear* —This point is a problem for the civilian and military outfitter but it seems unwise to make shoes water tight due to perspiration which leads to fluid accumulation unless some ventilation or absorption device is incorporated

(c) *Dryness* —Shoes and socks should be changed as soon as possible after exposure This can be done even under the exigencies of warfare and should be stressed to all officers and enlisted personnel Socks can be dried by canned heat the use of exhaust fumes from the motor the heat of a motor block or a small flashlight battery heater

(d) *Hygiene* —Since a major amputation often depends on whether infection is a complication or not cleanliness of the part before and after exposure is extremely important In the Korean area cold casualties in officers were relatively lower than those in the enlisted men despite the fact that the officers were older and suffered the same degree of exposure This emphasizes both self-discipline and hygiene In World War II the incidence of infected wounds decreased when all personnel were required to take a bath and put on clean clothing just prior to an invasion

(e) *Movement* —Movement of the parts is of primary importance Many of the worst frostbites which occurred in the Marines who were cut off near the Chanjin Reservoir in Korea were in those who had other wounds requiring them to ride out on transportation

(f) *Trauma* —Walking is a very important factor in secondary gangrene and if one must march after such exposure the incidence of gangrene will increase In World War II the amputation rate in the Navy in immersion

foot where there was no trauma was approximately 1 per cent, while the Army figures in the Aleutians where the trauma of walking was combined with exposure showed approximately 20 per cent

2. IMMEDIATE TREATMENT.—The wound should be classified as to degree of frostbite Further exposure to cold and wet must be eliminated. There should be no tightness or constrictive clothing Bed rest is important.

1. *General Treatment* —Antibiotic Drugs —Penicillin should be used empirically, any open wound is cultured and the antibiotic to which the organism is most sensitive is used

Nicotine —The patient should use no tobacco in any form. This is of great importance, as the relationship between smoking and the vascular complications of frostbite has not been emphasized sufficiently

Stimulants —Those in shock may need shock therapy. Whiskey is an excellent stimulator and vasodilator Hypoproteinemia, starvation and exhaustion all require specific therapy

Anticoagulants —In every case the anticoagulant drugs should be used to reduce the incidence of thrombosis unless other wounds preclude the use of these drugs

2 *Local Treatment* —The wound should be atraumatically cleansed. A G-11 preparation works well. If the wounds are infected, they should be covered with a sterile dressing Clean wounds may be left exposed, protected by a bed cage Sterile saline soaks for one hour a day remove any purulent or collected debris and keep the edges soft. Whirlpool baths may help

A *Slow Warming Method* —This method was based upon the fact that vascular damage in the frostbitten area is greatest in the venous and lymphatic vessels The pathology is compounded by the failure of these drainage systems to carry away fluid The arterial circulation, as it recovers, will bring increasing amounts of blood into the part. This blood supply is kept reduced by moderate cooling so that the amount of fluid brought in is not greater than the amount that can be carried away. Blebs, edema, discoloration and beginning necrosis are signs of too great a fluid supply. The injured part is kept in a tent which has a temperature of 55° to 60° F This temperature is maintained by icebags and electric fans together with coverings over the tent The therapy is continued for from three to five weeks During the latter part of the therapy the number of icebags is decreased until the part is at room temperature

The basis for these therapeutic ideas was supplied by the patients themselves during World War II They stated that they had been more comfortable back in the lifeboats than after they had been hospitalized. Some patients were observed to open the window and place the injured part outside

Such experiences resulted in the slow warming method

While the eventual place that slow warming will take in the therapy of those exposed to cold is not clear, the experiences with this method were sufficiently good for its application to be considered in each instance. There will be occasions always where the number affected or the lack of facilities require some type of treatment applicable to all the patients In such contingencies, the slow cooling will be the most effective method to

employ Under ideal circumstances some other therapy will replace this measure With a slow cooling method applied to some cold casualties from Korea the amputation rate in this group was extremely low In addition to the cooling regimen anticoagulant drugs were administered unless other wounds precluded their use Antibiotic therapy and *ultra conservative surgical measures* also were stressed

B *Rapid Warming Method* —Experimental work by Lange[42] Shumacker[41] Rosenfeld,[44] Radisch[47] and others demonstrated that in animals rapid warming of the part *but not heating* resulted in no more and at times less tissue loss than slow warming Shumacker's[48] later work showed that the tissue loss was small even if the part became frostbitten slowly as occurs in cold exposure The pathologic and physiologic reasons for these conclusions are not grounded too well History is replete with the gangrene following heating of frozen parts The experiences of Napoleon's army in the retreat from Moscow[43] the observations after exposure in the Klondike days the soldiers at the Bulge and in the Aleutian Islands all are examples In addition the inhabitants of our Northern states and Canada have prevented necrosis from the occasional unavoidable frostbite in those areas by slow warming and even applying snow It is of interest that the early reports on the use of rapid warming for this condition were received from the Red Army of Russia

These animal experiments however cannot be discounted[44] The rapid warming technic has a definite place in the treatment but its evaluation requires more experience It has been adopted by the American Red Cross and is advocated as the treatment of choice by the author This means *warming* but *not heating* the frostbitten part This can be accomplished with mildly warm water

Pressure dressings such as plaster casts have no place in frostbite therapy[46] Antihistaminic drugs such as Benadryl in combination with Rutin, experimentally appear to increase the salvage rate of the rapidly thawed limbs.[41] Large doses of ACTH have had no effect on the incidence of gangrene in the rapidly warmed part[44] Blockage of the sympathetic system apparently does not reduce the amount of tissue loss in the experimental animal[42] It is evident, therefore that the true place of rapid thawing in this therapy has not been clarified This measure however does have value

C *Other Treatment* —Other therapy of a general type includes efforts to combat infection with antibiotic drugs the use of anticoagulant drugs unless contraindicated by other lesions and conservative surgical principles These are detailed under the treatment of Trench Foot and Immersion Foot on pages 280 to 282

3 TREATMENT IN THE INTERMEDIATE STAGE (*Second to Twelfth Week*) — This period is of utmost importance to the patient. The management should include *sterile dressings exposure* of the part to the air in a tent, continuation of the *antibiotic* and *anticoagulant* therapy until the end result is established The most important point during this stage is to *avoid surgical intervention* Many limbs have been lost due to early or drastic operation The part may appear to be black and to the inexperienced hopelessly lost In a few weeks however it may be seen that the gangrene was superficial

involving only the skin or superficial tissues These may be shed by the patient, leaving healthy granulations underneath At times, these tissues come off like a glove or sock, leaving a viable extremity which will heal or can be grafted Time should be allowed to pass before any definitive surgery is performed Any infected areas which localize must be drained during this stage The use of sterile saline soaks may speed the separation of the dead from the live tissue and also aid in evacuating any secondary purulent collection To such solution can be added a sulfonated detergent such as pHisoHex with Hexachlorophene In some instances, such soaks are more beneficial when applied in a whirlpool type bath

*Sympathetic Interruption* —In some patients where the degree of arterial supply seems deficient, a block of the sympathetic nervous system may be of value If improvement follows, a sympathectomy may be performed This type of therapy must be individualized and should not be a routine affair Medical interruption of the sympathetics has helped some patients in this category The use of Priscoline, tetraethylammonium chloride or one of the intravenous anesthetics helps in selected instances (See pages 490 to 494 ) A drug, Oranixon (3-o-toloxy-1, 2-propanediol)* has, been useful in many of these patients, particularly in the relief of pain In one group so treated the patients were more relieved of their discomfort with this drug than with opiates This drug acts as a smooth muscle relaxer, has anesthetic qualities locally and may be adrenolytic in its effect (See pages 164 to 174 )

*Ambulation* —Walking should be restricted until the circulation is re-established Some patients must be treated as if they had arterial obliterations (See page 493 )

4 DEFINITIVE OR FINAL SURGICAL THERAPY. (*a*) *Amputation or Plastic Repair* —The question as to whether amputation or plastic graft will be the end stage must be decided by a team of the plastic surgeon, the surgeon who will amputate, and the director of the service The value of surgical conservatism is indicated by a record of only two major amputations in over 250 frostbites in one naval hospital. In World War II, many of these patients would have lost their limbs It must be stressed that any part of a patient's own limb that he can stand on is so far superior to the best prosthetic appliance available or contemplated that there is no comparison If we are willing to give time to tuberculosis and psychiatric care, and even to the hopeless carcinoma patients, these individuals are entitled to all the time necessary to resolve their lesions Some patients can be restored to function with split or full thickness skin grafts

(*b*) *Causalgia* —The incidence of causalgia is small but there is a definite number of patients who have this complaint as a residual of their exposure If this trouble persists, and a sympathetic block relieves the symptoms, sympathectomy may be performed (See pages 507 to 510 )

(*c*) *Hyperhidrosis* —This lamentable lesion develops in many of these patients If it does not respond to conservative and hygienic measures, sympathectomy offers hope for relief

**Trench Foot and Immersion Foot.**—ETIOLOGY.—Trench foot and immersion foot are military and naval counterparts of frostbite Trench

*Organon, Inc , Orange, New Jersey

foot occurs when the limb is cooled or wet and the part is subjected to trauma Immersion foot occurs when the limb is cooled or wet and the part is subjected to undue exposure, prolonged dependency and malnutrition With starvation in both conditions there is hypoproteinemia, and edema will result due to protein loss and because of the dependency alone

Trauma must be avoided in all cases of trench foot and immersion foot The degree of trauma determines the amputation rate The amputation incidence was approximately 24 per cent in trench foot with the trauma of walking The amputation incidence in immersion foot where the cooling dependency and malnutrition were not complicated by trauma was approximately 1 per cent

Symptoms.—The symptoms and signs of immersion foot are those of frostbite with the addition of edema due to dependency, malnutrition with wetting In trench foot the signs are the same but are aggravated by the trauma of walking The symptoms vary with the degree and length of time of exposure the presence or absence of infection the degree of trauma or dependency and the type or treatment rendered primarily Trench foot has been an important factor in warfare since early times The classic description of Napoleon s surgeon Larrey cannot be improved Larrey described the numbness and coldness in trench foot as a feeling that the feet are made of wood All symptoms are made worse ' when the boots are removed the picture then being complicated by swelling When the part is warmed there is an increase in swelling hemorrhage and bleb formation This is followed by local necrosis or gangrene Whenever the condition is complicated by the trauma of walking the necrosis is always more severe

The appalling condition was brought to the world s attention during the Battle of the Bulge in Belgium when there was no opportunity to change shoes or stockings the weather was cold and there were repeated exposures to water The Germans had fewer chances to change This may have been an important factor in our winning the war Thus the fate of wars and of nations may rest upon the control of this problem How important this may be is accentuated by a universal feeling that the next war will be fought in the Arctic areas

Treatment —The treatment of trench foot and immersion foot follows that outlined under frostbite with certain variations due to the military or naval situation at the time the facilities available and the presence or absence of other wounds The therapy may be divided into the prophylactic measures discussed on pages 276 to 278 certain general measures anticoagulant therapy and the early and late definitive surgical treatment

*Prophylactic Measures* (see page 276) —The prevention of so-called trench foot and immersion foot rests upon avoiding exposure to cold and wet keeping the part mobile changing the shoes and stockings as often as it is practicable and constructing windbreaks Early treatment must include removal of the constricting foot gear The rest of the prophylactic measures were detailed under Frostbite pages 276 and 278

The *general therapy* has been detailed under frostbite It includes the antibiotic drugs the abstinence from nicotine the use of anticoagulant

drugs unless other wounds contraindicate their administration Blalock showed that it required a 10° to 15° lower temperature to freeze limbs of animals when anticoagulants were administered than in control animals.[45] In patients who are shocked, treatment of this condition becomes primary. Starvation, exhaustion and hypoproteinemia must be corrected.

The *local therapy* has already been detailed. It consists of cleansing of the wound, sterile dressings, sterile saline soaks and whirlpool baths.

Whether the *slow warming* or *rapid warming* method of treating these patients environmentally should be selected will depend upon local conditions. Animal experimentation seems to indicate that rapid warming causes no greater tissue loss and perhaps less than slow warming. Recent results indicate warming *but not heating* the part is the best therapy.

The *surgical treatment* requires judgment. It could be best summarized by the words "surgical restraint." In the absence of uncontrolled infection, amputation never should be performed. Black necrotic areas frequently are superficial and shed off like a snake's skin, leaving viable tissue which will heal or which may be grafted. (See page 280.)

Some observers have advocated early sympathetic interruption in frostbite. Should the patient be seen prior to the thrombosis stage and respond well to a block, a sympathectomy may be advisable.

A sympathectomy may be indicated also to relieve the subsequent paresthesias and anesthesias after the decision of life or death of the part has been determined. The relief from hyperhydrosis, which so frequently follows such exposure to cold, may be sufficient reason for performing a sympathectomy inasmuch as these symptoms are extremely annoying to the patient and to others. For complete details of the treatment of these lesions in stages see pages 276 to 280. In like manner, causalgia may require sympathectomy.

## PERNIO (CHILBLAINS)

The first description of pernio was by W. Muller, who, in 1680, wrote a monograph called "De Pernionibus."[71] The vascular nature of pernio has been re-emphasized by McGovern and Wright.[70]

**Etiology.**—Pernio usually appears on the extremities and is associated with exposure to cold and wet weather. The lesions appear in acute and chronic form, the chronic condition following frequent exposures to cold and dampness. Pernio occurs in those susceptible to such exposure and does not occur in all those so exposed.

**Symptoms.**—In the acute stage of pernio, the skin is reddish or cyanotic with some edema. Small blebs or purpuric spots develop, and the pigmentation may remain for a long time. The acute stage of pernio is characterized by itching and a burning sensation which is aggravated by warmth. The acute stage usually subsides in fourteen days.

In the chronic state, ulcerative lesions may develop on the skin of the feet and legs. This occurs more often on the legs of American women coincidental with exposure of these parts by short dresses and sheer stockings. Wright contrasts this with the early German reports of rarely seeing the condition in the lower extremities. The German people were protected

by heavy underwear and stockings and high shoes Ulcers may persist throughout the exposure time during the entire winter The ulcers may heal again in the summer time but leave scars These healed scars frequently break down The skin often is cold and cyanotic New blebs may appear with superficial ulcers The severity of the symptoms depends upon the degree and the length of time of exposure

**Pathology** —The pathologic picture of pernio is not entirely characteristic but has three consistent findings

(1) Intimal proliferation thickening of the wall of the artery and periarterial and venous infiltration by lymphocytes monocytes and leukocytes

(2) Necrosis of the panniculus adiposus.

(3) A chronic inflammatory reaction in the subcutaneous tissue in which giant cells but not tubercles are found



Fig 83 —Pathology of pernio Section taken of the blood vessel in pernio as described by McGovern and Wright The section is not pathognomonic and similar changes are seen in some cases of acrocyanosis and trench foot

A similar pathologic picture may be seen in acrocyanosis livedo reticularis or trench foot

**Treatment.**—1 *Acute Stage* —While the reaction to temperature change has not been definitely ascertained in the light of our present knowledge the part should not be warmed suddenly or injured Keeping the part cool with ice bags for several hours helps Sterile dressings should be applied if there are any skin breaks. No salves or antiseptics should be used Exposure thereafter to cold and wet should be avoided Therapy essentially follows that advocated for frostbite

2 *Chronic Pernio* —There is no specific treatment for chronic pernio but some may respond to the treatment outlined under Cold Allergy (see page 284) A change in climate is not always satisfactory A northern climate tends to keep the condition active.

3 *Surgical Therapy*—If sympathetic nerve blocks indicate a good clinical response, a sympathectomy may be recommended Exposure to cold thereafter still may cause ulcerations. Prophylactically, in those susceptible to pernio, a sympathectomy may be of help

## COLD ALLERGY

Certain patients develop an intense and severe allergy when exposed to cold These reactions do not conform to the classification of the organic or functional diseases

**Etiology.**—These patients respond aggressively on exposure to mild or severe cold Some of them are troubled only in the winter by temperatures under −30° F Others have year around symptoms, being so sensitive that a cold wind, even on a warm day, causes allergic manifestations Temporary exposure in the more sensitive may be deleterious Some patients become so reactive that an air-conditioned or refrigerated atmosphere is dangerous for them These patients become acute problems with the modern tendency to standard conditioned temperature throughout the warm and humid months In some, the imbibition of cold drinks or food is sufficient to inaugurate the syndrome The application of a cold cloth or ice bag may precipitate an acute reaction Allergic manifestations have followed enemas or douches which are of lower than body temperature Shock has been precipitated by cold subcutaneous or intravenous injections There are cold, precipitable proteins in the blood in certain disease states These are seen in multiple myeloma, liver and joint diseases and a few in patients with Raynaud's syndrome That they may be present in other patients follows The critical temperature is 37° C Since the temperature of the extremities always is less than the rest of the body, the symptoms appear in these areas If the temperature of the blood is below 37° C, there is an abnormal collection of cells and a precipitate of cryoglobulin Warming of the part usually dissolves the precipitate [69]

**Symptoms.**—The symptoms depend upon the exposure, the precipitant, and the degree of sensitivity of the patient or part They vary from erythema or hive-like manifestations to a raised, thickened and indurated, local reactive area Muscle and joint changes have been reported as has been massive edema Edema of the glottis may be a serious problem in the oversensitive group It is difficult to draw blood from the veins of such patients

**Pathology**—The pathology again depends upon the exposure and the sensitivity Locally, there may be an infiltration of round cells and edema or signs of local inflammation Extreme sensitivity may resolve in massive and obstructive edematous changes with necrosis of the affected parts Sludged blood occurs in the more serious cases Continued exposure in non-vital areas may be followed by atrophy of surrounding structures

**Treatment**—(*a*) *Desensitization*—Slow but continued exposure to an increasingly cool stimulant may eliminate the allergic reactions Time and consideration are necessary to achieve results When the individual is a child, the mother may be able to overcome the sensitivity in months or in a few years

(*b*) *Histamine* —Since the reactions are similar to those provoked by histamine small doses of this drug increasing as the tolerance rises may correct the problem Since histamine itself is a strong stimulant overdosage at any time must be prevented

(*c*) *Others* —The other antihistaminic drugs such as pyribenzamine and benadryl may be tried and have proven effective in individual instances

(*d*) *Climate Change* —In extreme instances a change to a warm or less variable climate may be necessary The sensitive patient should be warned about and protected from cold wind wet weather and refrigerated areas One should be guarded in the prognosis in each patient.

## TRAUMATIC SEGMENTARY ANGIOSPASM (TRAUMATIC SEGMENTARY ARTERIOSPASM)

Blood vessels may develop a spasm as the result of trauma Various names for this syndrome have been suggested These include traumatic Raynaud s syndrome arterial stupor traumatic segmentary arteriospasm and traumatic segmentary vascular spasm Perhaps traumatic segmentary angiospasm best describes the condition since veins as well as arteries may be involved

Traumatic segmentary angiospasm has become increasingly important from a medicolegal standpoint with the broader interpretation of existing and new compensation laws relative to occupational diseases

**Etiology** —As the name implies traumatic segmentary angiospasm means a spasm of a segment of a vessel following sudden or continued trauma The trauma is not necessarily severe Such a lesion may develop in patients who traumatize their fingertips at work or on machines Often there is a history of past sympathetic nerve imbalance and nervousness We have seen the condition develop in comptometer operators who have used such a machine for twenty years The irritation of rubbing the fingers over fabrics prior to pressing them has produced lesions in the involved fingers of pressers Other arteriospasms may be due to chemical trauma ulcers have followed exposure to glue tar and dyes

Similar symptoms have been reported in grinders of small metal castings by several investigators.[72 77] The symptoms appeared about twenty-one months from the time they started this type of work In such grinding revolutions vary from 1 950 per minute to 3 050 per minute and the grinding is done in a circular like motion The workers hold the component in their hands tightly and press the casting against the grinding machine As they work on a piecework basis they become very agile in this operation

Thus many types of instruments may produce spastic changes in susceptible individuals That the instruments *per se* are not the cause is demonstrated by the fact that all individuals so exposed do not develop lesions

**Symptoms** —The lesions in traumatic segmentary angiospasm may vary from a mild spasm with blanching coldness and pain to ulceration infection necrosis and gangrene At times the symptoms are acute and

result in an acute arterial thrombosis In other patients, the symptoms are gradual in onset, with periods of increasing fatigue, coldness and numbness If the trauma is maintained in such patients, ulceration eventually will develop Many have a history of other allergies

**Diagnosis.**—A history of sympathetic imbalance, characterized by hypersensitivity to cold, may be a leading point to the diagnosis of traumatic segmentary angiospasm An organic constriction, caused by the pressure of a tumor or cervical rib, or by such occlusive arterial diseases as arteriosclerosis obliterans or thromboangiitis obliterans, should be ruled out by thorough clinical examination and roentgen rays

Capillary microscopy eliminates Raynaud's disease, and the oscillometric readings will differentiate organic occlusions The therapeutic test of removing the trauma, followed by improvement, is the diagnostic guide In some cases, arteriograms are necessary for a final diagnosis

**Differential Diagnosis.**—It is not difficult to differentiate traumatic segmentary arteriospasm from other vasospastic lesions, provided the condition is kept in mind Traumatic segmentary angiospasm may be differentiated from (1) Raynaud's disease, (2) thromboangiitis obliterans or arteriosclerosis obliterans, (3) Sudeck's atrophy, and (4) causalgia, by the following points

(1) *Raynaud's Disease* —The patient's occupation, the sex of the individual, the bilateral incidence and the underlying characteristics of Raynaud's disease (see pages 258 to 260) will be helpful in differentiation

(2) *Thromboangiitis Obliterans or Arteriosclerosis Obliterans* —A patient with traumatic segmentary angiospasm, as well as pneumatic hammer disease (see pages 287 to 289), may have symptoms similar to thromboangiitis obliterans and may progress even to gangrene Thromboangiitis obliterans occurs usually in the feet Efforts should be made to prove that the occlusive disease is present in other parts of the body In some patients a change of occupation with resulting improvement will be diagnostic as a therapeutic test Biopsy may be required

*Allen's Test* —The use of Allen's test[73] may be helpful in ruling out an occlusive arterial disease This test is based on the normal communication between the radial and ulnar arteries The patient clenches his fist tightly while the physician applies pressure with his fingers on both the radial and ulnar arteries The patient then opens his hand The physician's finger is then removed first from the radial artery The filling time in the hand in seconds is denoted by its changing from blanched white to normal pink This is then repeated for the ulnar artery In normal individuals, the hand will return to pink in approximately three to five seconds In those with occlusive arterial disease, the return to pink is considerably delayed

Lesions similar to thromboangiitis obliterans have been seen in patients using pressure instruments If the occlusive lesion appears while the patient is using an instrument at work, a complex medicolegal problem may arise [73] (See Cardiovascular Disease and Trauma, page 804 )

(3) *Sudeck's Atrophy* —The differentiation of traumatic segmentary angiospasm from Sudeck's atrophy should not be difficult, inasmuch as the bone changes are quite typical in Sudeck's atrophy (See page 296 ) The history of slight trauma followed by pain, swelling, anesthesia, limitation

of motion paraesthesia together with the roentgen ray signs are diagnostic for Sudeck s atrophy Some atrophy similar to a Sudeck s type occurs in angiospasm For specific therapy in such instances see pages 297 and 298

(4) *Causalgia* —The difference between traumatic segmentary angiospasm and causalgia may be one of degree only Many of these patients develop the syndrome known as causalgia When causalgia develops sympathectomy is the treatment of choice For details see page 502

**Treatment.**—The fundamental therapy requires that exposure to the causative trauma be eliminated In an industry this may be difficult as the individual may be specially trained and unable to perform other work for equal remuneration This disparity must be corrected however in each case as shortsightedness in this respect may result in a permanent total disability

Conservative treatment with moist warm dressings is of value Warm whirlpool baths mild massage and active and passive motion also is helpful

Exploration of the artery is not indicated In one series of 44 pateints so treated 6 major amputations resulted If gangrene develops amputation will be necessary

Surgical treatment should be ultra conservative since in the upper extremity even a small part of the phalanx may enable the individual to continue some type of work

## VIBRATORY PRESSURE DISEASE (PNEUMATIC HAMMER DISEASE SPASTIC ANEMIA MECHANICAL DRILL DISEASE)

The trend to use descriptive adjectives to define a syndrome seems logical and this title includes lesions formerly described as pneumatic hammer disease mechanical drill disease dead fingers spastic anemia pressure spasm and various other descriptive terms These patients are classified together because regardless of the cause or course their symptoms are similar

**Etiology** —This disease follows the use of instruments or machines in which there is a pressure exerted on the hands by the vibration and secondarily by the escape of cold air which drives the machine In respect to the causes they vary from the previously discussed lesion angiospasm

Vibratory pressure disease develops in stone cutters who use a pneumatic hammer Lesions occur in a large number of those exposed The original description of the condition under the name of pneumatic hammer disease was made by Cottingham[78] of the U S Department of Labor in 1917 Cottingham s study was undertaken at the request of the Stone Cutters Association of North America and was prompted by the high incidence of the condition among its craftsmen

An *air hammer* is an instrument which has a handle weighing from 3 to 6 pounds and is usually 10 to 12 inches in length The hammer is driven by compressed air at approximately 3000 strokes a minute. A chisel is inserted in the end of the handle This chisel is guided in righthanded individuals by the left hand The right hand holds the handle as one would hold a pen The fourth and fifth fingers of the left hand being nearest to

the end of the tool are pressed against it to guide it It is on the fourth and fifth fingers of the left hand in righthanded persons, and vice versa, that the disease is seen most often and earliest Since the cutters of limestone use the instrument more than persons in any other industry, it develops most often in this group

A similar condition has been observed in shoemakers who also use a vibrating machine

These air hammers differ from the instruments used in structural iron work where a much heavier instrument, delivering only about 25 strokes a second or 1500 a minute, is used Such a vibration rate does not cause the disease The disease is due to the vibration and escape of air under pressure Vibratory pressure disease is found in miners, stonecutters, shoemakers and riveters Steel workers whose drills vibrate at 1500 strokes per minute seldom are afflicted

**Symptoms.**—The symptoms of vibratory pressure disease develop in from two months to two years from the time the individual begins to use the pneumatic hammer, compression drill, air drill or air hammer The symptoms are noticed particularly in cold weather In one group of patients, 4 per cent developed symptoms in two years and 55 per cent noticed changes in from three to ten years after the beginning of that occupation

That the escape of cold air and vibration is a factor is evidenced by the failure of a similar syndrome to develop in occupations such as hod carrier or loader, where equal pressure is exerted on the hands

The attacks begin with areas of blanching and numbness These symptoms appear after work or in the morning

There are areas of anesthesia, a feeling of clumsiness, and some pain and discomfort The severity of symptoms varies with the individual and the degree of exposure, and, at times, a whole hand may be involved In some, only the right index finger and thumb have been affected

Calouses may develop in the skin, and there have been reports of changes in *muscles* and *joints*, with some gross muscle atrophy The joint symptoms are attributed to trauma and affect the elbow or shoulder joint There may be roentgen ray evidence of *calcification* of the *articular capsule*, and the head of the humerus has been found to be deformed

Relief follows cessation of the use of the instrument, and there may be no symptoms whatsoever between the attacks The secondary changes are not too well understood The same condition may recur as long as ten years afterward when the same type of work is resumed or when the part is exposed to cold Gangrene has been reported [82]

That the instrument itself is a factor in causing the disease is proven by the large number of patients who have this syndrome If patients with an underlying vascular disease, such as arteriosclerosis or thromboangiitis obliterans or those who are subject to spasm or frostbite, do this type of work, they have symptoms earlier and to a greater degree

**Pathology.**—The pathologic changes in vibratory pressure disease result from impairment of the circulation in the hands due to the pressure and vibration of the tool, and the escape of cold air

With the continued impairment of circulation, changes similar to Raynaud's syndrome develop The changes range from erythema, spasm and

pallor to ulcerations and gangrene. In over 65 per cent bone changes develop and roentgen ray examination shows bone cysts and osteoporosis in the majority of instances.

The possibility that the actual pathology occurs from a destruction of the capillaries by rapid and repeated trauma has been advanced.[81]

**Prognosis.**—When susceptible patients continue their occupation the prognosis is poor and amputation may be the end result. An occupational change is indicated.

**Treatment.**—The most important factor in the treatment of vibratory pressure disease is the employment change. This may be a difficult personnel and economic problem. It may be avoided prophylactically by eliminating from work of this nature those with a history of cold sensitivity ("poor circulation") and difficulty with previous similar occupations. Pre-employment questionnaires and physical examinations should include a check for this condition.

A thought for the future is the training of an individual for two different types of trade with a shift from time to time. This applies to all occupational disease problems.

Where applicable the use of pressure machines with vibrations of less than 2300 strokes per minute will lessen the incidence. A resilient pad to take up part of the vibration also may be a prophylactic aid.

Physiotherapy is of no therapeutic value in vibratory pressure disease. The only treatment necessary in vibratory pressure disease is prophylaxis. Patients with this disease should be transferred from their occupations as soon as they are discovered.[79–82] Surgical therapy may be necessary for ulcerations and gangrene.

## GLOMUS TUMOR
## (ANGIONEUROMYOMA, GLOMANGIOMA, ANGIOSARCOMA)

Glomus tumor was first described by Wood in 1812.[83] It has been designated glomangioma or arterial angioneuromyoma.

**Etiology.**—The underlying anatomy demonstrates the nature of glomus tumor.

In order to provide large amounts of available blood when there is a sudden need for it, the body contains many arteriovenous shunts, so-called glomi. The skin of the hands, for example, has innumerable glomi. These glomi, lined with endothelial cells, are adequately enervated by sympathetic nerves so that reflex dilatation and adequate blood supply are available automatically when needed. In the normal individual there may be four or five hundred such glomi per square inch of body skin surface. These vessels have large cuboidal endothelial cells lining their lumens and a muscular layer. About this layer there are large epithelial cells with oval nuclei called "glomus" cells surrounded by nonmyelinated nerve fibers. Around these nerve fibers are the veins.

These epithelial cells may develop benign tumors and with growth these tumors exert pressure on the nerves and blood vessels. External pressure on such a tumor results in a spasm of that part with severe pain. This is the condition known as glomus tumor. Tumors occur at any age

There is supposed to be a relationship to trauma. Since these injuries so often occur on the fingers it is difficult to attribute the tumor to one specific injury. Trauma may bring an already existent tumor to the patient's attention.

**Symptoms.**—Glomus tumors develop most frequently under the digit nail. On serial section of 64 suspected toes, Popoff[65] found glomus tumors in 24 under the nail.

Usually, glomus tumors are the size of a pin head and, because of their vascular component, have a bluish discoloration. Pressure on glomus tumor areas causes blanching and spasm of the part, and a pain of such severity that it has been likened to that of a foreign body in the eye.

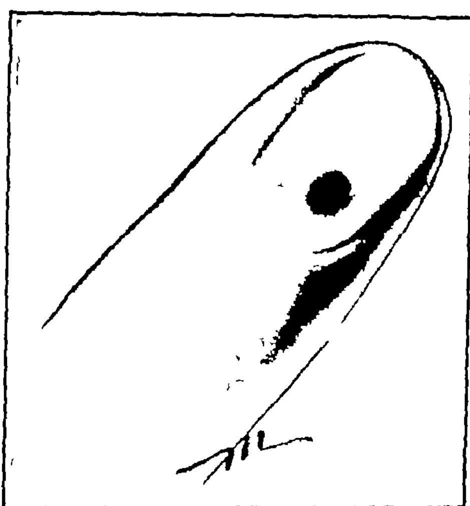


FIG 84.—Glomus tumor under the fingernail. This is a frequent site of such glomus tumors and causes many symptoms because of the exposed position of the tumor.

The tumor may be so small that its color is masked. Often the patient has been treated for arthritis, neuritis and, unfortunately, sometimes for neurosis. The tumors are usually single but sometimes multiple. The cardinal symptom is the bluish, discolored area which upon pressure causes severe pain and spasm.

**Diagnosis.**—The diagnosis is not difficult if the examiner has ever seen a glomus tumor or remembers its existence. The pain can be produced by exposure to cold. Other tumors rarely cause difficulty. The hemangiomas, neurofibromas, or angiosarcomas are not painful. Pathologic differentiation is the final point in diagnosis.

How often glomus tumor is not diagnosed is illustrated by the fact that three physicians taking postgraduate courses in cardiovascular surgery, given by the author, have presented themselves as patients with such tumors, the diagnosis having been made by themselves while listening to the lecture.

**Pathology.**—Glomus tumor is a hypertrophy of the normal glomus structure heaped up and multiplied. The characteristic pathologic picture shows the large glomus cells with hypertrophy of the muscles around the blood vessels and usually an excess of nerve tissue. There is no evidence

that these tumors metastasize although there is a local invasive tendency[81] One of our patients developed a glomus tumor after cutting her finger in the laboratory A painful syndrome developed which was unrelieved by excision nerve crushing sympathectomy and amputation This condition spread to the third finger There is no evidence that these tumors metastasize but local spread is possible

**Treatment.**—Complete surgical excision is curative Efforts to use roentgen ray and radium therapy have been ineffective and all other conservative measures fail to bring relief Surgical excision should include the periosteum of the bone and if sufficiently radical a cure may be expected in each case Some mutilation of the finger may result It is best to excise the matrix of the nail so that there is no danger of leaving any tumor cells

## CAUSALGIA

Causalgia is a syndrome characterized by burning pains trophic changes and recurrent spasms which follow an injury of or in close proximity to a peripheral nerve It has been known for a long time that relatively minor injuries can initiate this syndrome characterized particularly by pain and this fact has interested all surgeons The description of this syndrome as originally made by Mitchell Morehouse and Keen[82] from their experiences in treating soldiers in the war between the States in 1864 cannot be improved They defined causalgia as hyperesthesia of the hand or foot following an injury in the region of a peripheral nerve

The condition was at first believed to be primarily neuritic but the important role that the blood vessels play was pointed out by Leriche and Fontaine[83] in 1935 Albert[84] in 1930 described the relationship of the condition to joint injuries

**Etiology**—Causalgia often occurs after an injury which may not have been a severe one The symptoms are out of proportion to the degree of injury

Some investigators have tried to attribute the condition to irritation of special trophic nerves Lewis[85] in 1937 stated that causalgia arises from an unrecognized set of nocifensor nerves and he was of the opinion that the stimulation of these fibers lowered the threshold of the ordinary nerve apparatus for pain.

Leriche and Fontaine[83] in 1935 described the condition as probably due to abnormal vasomotor reflexes

White and Smithwick[86] summarized the present status of the etiology of causalgia by stating that very little is known about the cause of this condition There is extreme pain accompanied by an abnormal vasomotor activity and response and the cause often is a comparatively mild injury

**Symptoms**—The *pain* varies from a mild or burning type to one in which the patient has a constant distress of extreme degree This pain may be excited by mild palpation or may be initiated by emotional or thermal stimulations The patient is hypersensitive about the affected part and protects it constantly to prevent someone from touching it According to White and Smithwick,[86] the nerves most commonly involved in causalgia are the median and the sciatic.

Symptoms other than pain include *trophic changes*, and *vasomotor instability*. The skin of the involved part may be bluish or red, and most often is cold and perspires freely. More rarely the skin becomes shiny, scaly and dry. The atrophy that occurs is partly due to disuse and partly on a trophic basis. The skin may lose all of its hair, and there is ridging, atrophy and breaking of the nails.

The *vasomotor changes* are due to a marked disturbance in the circulation of the part. Early in the condition, there appears to be more vasodilatation, but as the condition continues, there is vasoconstriction. Parts of the body other than that which is injured may also be involved.

The patients are usually of the nervous and emotional type, they become high strung, hyperesthetic, and some even verge on the psychotic stage. In some patients, addiction to drugs or alcohol is part of the picture.

The condition may arise in an amputation stump, where it is called "phantom limb" pain. (See page 247.)

**Pathology.**—Usually in causalgia there has been an organic injury to the part, and this is most often close to a peripheral nerve. There is cellular infiltration of the subcutaneous tissues, with secondary atrophy and sometimes contracture and pathologic evidence of wasting. In Mitchell's and his associates'[92] report, in 1864, there were many examples of painful lesions in the nerve trunks. Induration of the subcutaneous tissues is of the brawny type with fibrosis, and there have been reports of collagen deposits around the nerves.[94] In causalgia after an amputation, there may or may not be a neuroma of the severed nerve end.

**Diagnosis.**—The diagnosis of causalgia is not too difficult when the symptoms described above are present. Too often the patient is considered as neurotic. Sometimes causalgia occurs in an amputation stump after a major amputation. In such cases, the psychogenic factors for the pain must be ruled out. Where the problem is complicated by an insurance or compensation claim or by the refusal or inability of the patient to return to work, the solution is more difficult. At times, the diagnosis is made as the result of improvement under a certain type of therapy specific for the particular ailment. Accusations of cowardice and court-martials in military surgery have resulted from lack of understanding of this lesion.

**Treatment.**—Since there is such a marked vasomotor reaction in true causalgia, treatment has been directed toward interruption of the sympathetic pathways by any of the following methods:

1. *Repeated Nerve Blocks.*—Repeated sympathetic nerve blocks may give a complete cure. Where the ganglia have been blocked for a sufficient length of time, the painful synapses may not reform. The success of sympathectic nerve block treatment for causalgia can be determined by whether the patient receives relief with the first block and whether this relief persists for over four hours. Each subsequent block should produce more prolonged relief.[55]

2. *Surgical Sympathectomy.*—If the nerve blocks relieve the symptoms, sympathectomy will help the patient. The sympathectomy operations have been described on page 498. These procedures are effective in those patients who do not have a psychogenic basis for pain. The variability of the results in thoracic sympathectomy must be remembered.

3 *Medical Sympathectomy* —The use of the chemicals such as tetraethylammonium chloride Priscoline and intravenous procaine etc may have their best application in this group of patients They should be tried prior to any of the other measures.

4 *Periarterial Sympathectomy* —The periarterial operation as described by Leriche[30] in 1913 has been abandoned White and Smithwick[14] suggested that any success from this procedure is due to the general cutaneous hyperemia which follows the destruction of tissue in any surgical incision and is not due to any effect on the circulation or pain In this way,' they state the effect resembled the procaine block or foreign protein shock although it is of considerably greater duration Another operation may initiate more causalgia Better methods exist for denervating the sympathetic system

5 *Reamputation Resection of Neuromata and Plastic Operations* — These measures on painful amputation stumps usually fail to relieve the pain unless there is organic pressure on a sensitive area or a neuroma

6 *Rehabilitation*—A *rehabilitation* program is most important for these patients and should not be neglected *Physical therapy* may be of value after the initial problem is relieved Patients with causalgia fear that the pain will come back Many times they develop marked atrophy because of disuse of the part as a protective mechanism to keep them from going back to work. A graded exercise program and resumption of activity is of fundamental importance

7 *Other Surgical Treatment* —Sectioning of the posterior roots, severing of the spinal thalmic tract in the medulla oblongata and the intraspinal injection of alcohol have been tried[17] but these are serious operations and may be considered obsolete

8 *Bone Replacement Therapy* —There may be marked atrophy of bone due to disuse or associated Sudeck s atrophy Calcium and parathormone replacement therapy may be required (See page 298 )

*Summary* —In conclusion in the management of patients with causalgia the physician must first eliminate neurotic causes for the pain. The compensation or insurance angle must be investigated Malingering should be excluded In a true causalgia the interruption of the sympathetic pathways offers the best chance for relief of the symptoms If this operation is followed by an adequate rehabilitation program most of these patients can be restored to health and brought back to a gainful occupation The use of drugs like tetraethylammonium chloride or bromide Priscoline or Dibenamine are worthy of clinical trial although their efficacy and the dosage schedule are still doubtful *Psychotherapy* may be necessary to correct the accompanying psychoneurotic symptoms which have developed as a result of the pain

Operatively surgical sympathectomy is the treatment of choice Periarterial sympathectomy is ineffective and may in itself initiate causalgia or psychoneurotic changes if the latter operation fails

In treating any case of causalgia it should be borne in mind that there are certain allied conditions similar to causalgia which follow injuries to nerves blood vessels ligaments and joints. Among these are traumatic arthritis and rheumatoid arthritis and other lesions similar to those in which there are vasomotor and pseudomotor disturbances In these cases

if there are associated sympathetic symptoms, sympathectomy may be indicated The replacement for atrophic bone has been discussed

## HYPERHIDROSIS

**Definition.**—By hyperhidrosis is meant excessive sweating or perspiration

Sweating is a normal function of the sweat glands of the entire body Perspiration is a physiologic process following exposure to excessive heat and helps to eliminate heat and balance the body's temperature There has been considerable difference of opinion over which nervous centers control sweating since Langley[101] (1891) described the nerve fibers in sweat glands It is believed that the sweat glands are enervated by the sympathetic system The center most likely is in the hypothalmus which regulates the rest of the body temperature [98,101] The sweat glands act reflexly as a result of thermal stimuli They may respond also to emotional stimuli The sudden cold sweat of fear is such a reaction The reflex is specially active in competitive sports or public presentations It has been noted in race horses and in dog shows [94] This is a normal cooling reaction and makes the body aware of the approaching need for tension

There is another type of abnormal perspiration which affects particularly the hands or feet or both In individuals so affected, there is periodic or continuous over-sweating of the hands or feet One medical student could not assist at operations because of perspiration constantly running out of his rubber gloves [94] One of our patients had to buy 36 pairs of shoes a year because the excess moisture of her feet caused them to deteriorate Another patient had so much perspiration of a malodorous type that he had to be restricted from athletics because his companions could not tolerate the stench in the dressing room The psychic insult to the individual is great This lesion has caused suicides

**Etiology.**—There is no definite etiologic factor that causes hyperhidrosis, but it is due to hyperactivity on the part of the sympathetic nervous system This action is stimulated by nervousness, excitement, emotion, fear, or in some, by mild thermal or chemical stimulation In some patients, the condition becomes self-perpetuating The patient develops an anxiety state regarding the perspiration so that it may appear at any time, the anxiety being the stimulant to the perspiration symptoms The sweat glands are distributed over the entire surface of the body. The ones in the axillary and inguinal regions are larger and the number of glands varies The human averages 2,400,000 sweat glands according to Krause [99] There are more glands per square inch on the palms of the hands and soles of the feet than in any other part of the body

**Symptoms.**—The primary symptom of hyperhidrosis is excess perspiration, in which the extremities, in particular, become moist and cold This perspiration is usually limited to the palmar or plantar surfaces of the fingers or toes It may be normal above the wrists and ankles

The disability becomes extreme It has interfered with many patients' normal work and caused some to move to cooler climates Many of the patients become recluses, embarrassed by their ailment, particularly by the severe odor when the hyperhidrosis is in the feet

**Pathology.**—The pathologic picture of hyperhidrosis depends on the extent of the disease. The sympathetic nervous system is hyperactive to stimuli to which the patient is susceptible, i.e., tension, cold, heat. The sweat glands hypertrophy and multiply in number. Edema is present.

**Treatment.**—1. *Medical measures* have not been too successful. The use of formalin and other substances of this kind to harden the skin have caused maceration and irritation. Chronic irritation has followed the efforts to radiate these areas.

(*a*) *Deodorants.*—In the light of our present social status, excess perspiration or the odor thereof is an important problem. In America, body odor is a hygienic error which cannot be accepted or tolerated. Since the muscular effort of work or play results in such perspiration, efforts to control it have developed.

Such drugs as aluminum chloride, aluminum aceto-tartrate and zinc peroxide have been used individually and in conjunction to neutralize these odors. At times these have been combined with some drying or astringent agent such as alum, which has as its purpose to prevent perspiration at the area. Neutralization is more desirable than elimination of perspiration. These substances are used generally throughout civilized countries and some twenty different formulas and combinations are manufactured under various trade names in the United States. These are now in the same category as powder, toothpaste and creams as part of the necessary toilet accessories. In mild cases of hyperhidrosis the problem is solvable in this manner provided sufficient baths and local cleansing are combined for the basic hygiene. The importance of the perspiration problem in all classes of people slowly is becoming recognized, but it is to be emphasized that no chemical can replace the fundamental body cleanliness achieved with soap and water. The sulfonate detergents play an effective part.

(*b*) *Chlorophyll and Its Derivatives.*—A plant derivative, chlorophyll, which has to do with plant metabolism, has been reported to have a beneficial effect on healing for many years. Its local action in eliminating the odors of wound drainage led to its utilization through ingestion to combat body odors. Thus, we have forms of chlorophyll which appear to have an effect on the odor of perspiration. The ingestion of lozenges of chlorophyll allegedly counteracts odors of alcohol, tobacco and offensive food odors such as garlic and onions. Most of the commercial preparations available are in the form of tablets containing 100 mg. of chlorophene, which is the equivalent of 10 mg. of chlorophyll. The possible detrimental effects of overuse have been suggested. Recently studies on this substance as a deodorant against such diverse gases as formed by skunks, onions, garlic and scents as well as perspiration were reported as failures. The gases were not eliminated nor were they lessened by the ingestion of or mixture with chlorophyll.[97]

(*c*) *Adrenolytic and Sympatholytic Drugs.*—The use of adrenolytic and sympatholytic drugs is of value and should be tried in all mild cases. Many patients so treated in the early part of the disease have cures or long relapses. Priscoline particularly has had good effects and has less untoward reactions. Tetraethylammonium chloride causes varying reactions in different individuals. Dibenamine, in its present form, may be dangerous.

2. *Surgical Therapy*.—For extensive hyperhidrosis in the lower extremities and in those patients not amenable to more conservative measures, surgical sympathectomy is the treatment of choice. The excellent work of Roth[103] as well as that of Ray,[102] which showed the areas of nonperspiration that occur after various operations on the sympathetic nervous system, has been of value as a guide to sympathectomy for the relief of local sweating. Braeucker reported the first sympathectomy for hyperhidrosis in 1928.[96]

The operative technic has been well standardized and is described in the chapter on Interruption of the Sympathetics, page 498. If this operation fails one must consider that it may be surgical technic, inasmuch as the sympathetic nerves control the activity of the sweat glands, and once these are interrupted, sweating ceases. In no other condition is the relief as spontaneous and as satisfactory as it is in the treatment of lower extremity hyperhidrosis by sympathectomy. In the upper extremities, a satisfactory sympathetic denervation is more difficult and makes the results more uncertain. Surgical sympathectomy, therefore, for sweating of the hands may require both a pre- and postganglionic denervation.

## ACUTE BONE ATROPHY, ACUTE OSTEOPOROSIS, ACUTE BONE DYSTROPHY, SUDECK'S ATROPHY

This syndrome was described in 1900 by Sudeck[114], as an acute atrophy and osteoporosis occurring particularly after trauma. Sudeck originally considered it as a vasospastic lesion and differentiated it from the osteoporosis seen in atrophic disturbances associated with certain diseases of the central nervous system.

**Etiology**.—The etiologic factors responsible for Sudeck's atrophy are not too well known. The disease occurs after a mild trauma and it may accompany disuse. The atrophy develops much more rapidly than that attending normal disuse of a part. It may occur even while the part is in use.

There are two theories as to the cause of Sudeck's atrophy. One is that the condition is the result of reflex action through the vascular system as the result of trauma. The second is that the osteoporosis is due to inactivity or failure of normal stimulation to the bone. The vascular factor seems the most important one in the development of osteoporosis after trauma.

The suggestions that the lesion is due to a vitamin deficiency or simple calcium loss have not been substantiated. Parathyroid dysfunction may be a contributory factor.

**Symptoms**.—The symptoms of Sudeck's atrophy are usually in the bone. Osteoporotic changes occur, in which there is spotty atrophy, loss of calcification, and absorption of the bone from the part. This follows an injury which may not have been severe. With these bone changes, there is usually swelling, edema and coldness of the part with venous stasis. There is extreme tenderness and, at times, a causalgic-like pain. The part may be cold and yet perspiring. At times, there is so much bone absorption that fractures have occurred. The overlying tissue becomes red, swollen and tender. Disability is progressive and extensive.

**Pathology**.—The pathology of the bone change has been described above. There are also secondary soft tissue edema, venous congestion and arteriolar spasm. Atrophy of the soft tissues follows.

**Treatment**.—1. *Medical Measures*.—This type of treatment has not been too satisfactory. It is necessary for the patient to use the part and weight bearing should be encouraged. Physiotherapy in the form of active and passive motion, massage and heat may help. Cod liver oil and calcium lactate have been administered. Psychotherapy may be required in the atrophy of disuse.



Fig. 85.—Sudeck's atrophy. X-ray taken three months after a minor injury to the foot showing marked atrophy of all the bones of the ankle and foot. There was superimposed soft tissue swelling. Treatment was sympathectomy and the use of parathormone and calcium by mouth.

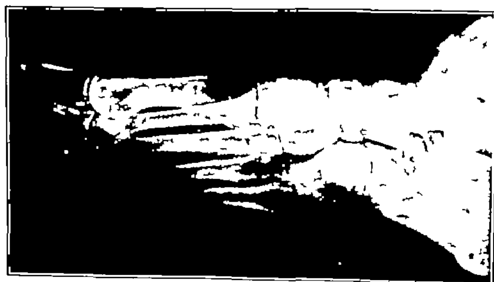


Fig. 86.—Second x-ray shows appearance of the same foot as Fig. 85 two months after therapy. Patient of Dr. A. F. Angelo. X-ray courtesy of Dr. Lucille Bond.

2 *Surgical Therapy* —Sympathectomy has been of great benefit in selected cases In most instances, the symptoms have been relieved immediately following sympathectomy Where there are no psychoneurotic changes, a good result may be expected. Sympathectomy should be of the complete type, such as that described in the chapter on Interruption of the Sympathetics, page 498

*Calcium and Parathyroid Therapy for the Bone Absorption* —In certain patients, sympathectomy alone is not sufficient Replacement of the calcium in the osteoporotic areas may be necessary for recovery In many patients after sympathectomy, the roentgen ray appearance of the bone has been restored and residual symptoms eliminated by supplying calcium and parathyroid hormone in the following dosage· $\frac{1}{10}$ gr parathyroid 3 times a day and 2 capsules of dicalcium phosphate with viosterol $7\frac{1}{2}$ gr, 3 times a day

Parathyroid will help in the mobilization and the deposition of calcium. The parathyroid, if continued, will cause absorption of the calcium and it should be discontinued after ten days

These two preparations have been of great help in the treatment of the resistive types of osteoporis or Sudeck's atrophy

## ARTERIOSPASM SECONDARY TO VENOUS LESIONS

That gangrene can develop without organic arterial occlusion as a result of venous disease has been known for a long time According to Haimovici,[113] Hildanus[114] recognized such a possibility as early as 1593

Vascular specialists have seen the condition either to a minor degree or to a point of actual gangrene The subject has been discussed by many writers including Cruveilhier[111] and Buerger in 1924[110] and was produced experimentally in 1937 by Fontaine and de Souza-Pereira [112] While gangrene without organic arterial occlusion is rare, it can originate from a venous stimulant by reflex spasm alone

**Etiology.**—The arterial spasm directly follows a venous thrombosis which often is of the massive type The veins are well supplied with nerves and these may initiate the reflex spastic mechanism which causes the artery to constrict The mechanism may be the result of liberation of a chemical substance which starts the arterial spasm. There will be venous spasm at the same time In many instances the arterial pathology appears to be due to the venous congestion being so great that the edema, infiltration and swelling mechanically obstruct the arteries The mechanical obstruction may be more important in the pathogenesis than spasm An iliofemoral thrombosis or thrombophlebitis at times causes enough reflex arteriospasm to temporarily or even permanently occlude the artery This occurs most often when there is a massive venous thrombosis which includes most of the collateral veins The afferent stimuli from these venous clots initiate efferent sympathetic activators which initiate both venous and arteriospasm if one subscribes to the neurogenic theory of spasm If the reflex is chemical some stimulus such as adrenalin or spastin is secreted into the blood stream and causes a spasm A case in point is given on page 300 and figure 87

**Symptoms** —The symptoms are those of interference with the superficial circulation to which is added edema primarily with increased heat and later with coldness of the part At first the major pulses will be felt, but with edema these will soon be obliterated The gangrene may appear at first in one or two toes which spreads to the other toes or even to the dorsum of the foot If these patients are treated conservatively long enough it is found often that most of the gangrene is of a superficial nature and does not require a major amputation

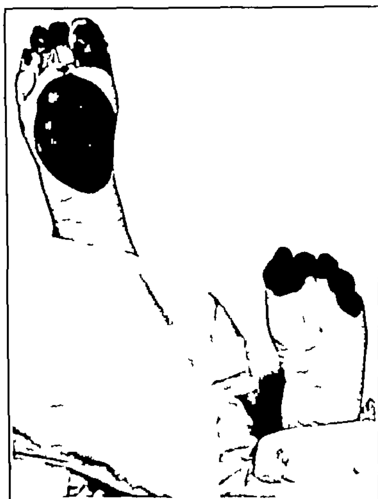


Fig 87 —Arteriospasm of right leg secondary to venous thrombosis. Intense edema followed by gangrene. Note opposite leg had arteriosclerotic occlusive disease. Courtesy Dr T Carey

**Pathology** —The pathology is one of a massive venous occlusion including not only the major vessels but the collateral vessels. If the arterial supply continues at an undiminished rate more fluid is pumped into this limb than can be removed As a result, edema develops and secondarily to this spasm and bleb formation With more edema there is more restriction to the arterial supply and merely by a mechanical nature the arterial circulation may be shut off by pressure To this is added the reflex arteriospasm which is of secondary importance to the development of the necrosis

**Treatment.**—This therapy is dependent upon the stage in which the manifestations appear. Treatment of these conditions, in general, should be conservative. One should prevent first the arteriospasm if that is possible and certainly propagation of the intravenous clotting should be prevented. Drugs of a ganglion-blocking type may be helpful. Anticoagulant therapy is extremely important and should be used continually at a therapeutic level until the result in the extremity has been decided. Early amputation is not advised and demarcation should be permitted to completion in the absence of a spreading infection. In most instances, the gangrene will be self-limited, somewhat like that in frostbite. The occluded superficial skin and subcutaneous tissue may be shed like a glove with fresh granulations below which may heal spontaneously or can be grafted. Sympathetic nerve blocks are helpful in some instances. Sympathectomy has been performed with beneficial results. The part is unable to remove the fluid at the rate it is brought into it and both the venous and lymphatic components are overworked and blocked. If the edema is increasing, the part should be elevated enough to control it. When it is controlled slight dependency helps. Warm packs are valuable. In some patients cold applications have controlled the massive swelling. These measures depend on the state of the patient and necessarily must be individualized.

*Case Report* (Fig. 87).—This patient with an extensive arteriosclerosis had had a cerebral accident. She was incoherent and incontinent with a hemiplegia on the *left* side. She developed necrotic lesions on the toes of the left foot and this foot was cold and there were no arterial pulsations present. The oscillometric readings were 0. It was apparent that she had an arterial occlusion on this side. This was treated by the use of perivertebral blocks, warm packs, lowering of the extremities, etc.

During the course of her disease, her *right* leg suddenly became edematous, swollen, bluish, quite warm at first and then cool. She then showed signs of congestion in the chest and *x*-ray evidence that there had been a small pulmonary embolus. The edema on this right leg rapidly extended, cyanosis increased, and soon the pulse was obliterated. Gangrene developed in the digits and partially over the dorsum. When the part was raised extremely high, the edema was reduced and there was a diminution in the extension of the gangrenous process. This was further improved by the application of ice packs. It was necessary, therefore, for the left side, in which there was an occlusion on an arterial basis, to be lower than normal, to be kept somewhat warm, and to be treated with the sympathetic nerve blocks. In other words, all efforts were made to increase the arterial circulation on that side. On the opposite side, however, there was gangrene originating in a massive venous occlusion. Too much blood was blocked in this extremity and there was reflex arteriospasm. When more blood entered the extremity, edema and more spasm resulted. It was necessary, therefore, to treat this right extremity by efforts to reduce the circulation to the part, *i.e.*, cold packs and high elevation.

## SPASTIC VASCULAR LESIONS DUE TO DRUGS, (EXPOSURE, INJECTION, INGESTION)

Some drugs and metals may cause vascular lesions when the patient takes them internally by injection or is exposed to them. Such drugs as ergot, adrenalin or ephedrine, or any of the by-products of these substances, have spasm-producing qualities. The heavy metals such as arsenic or

lead produce specific lesions Other drugs may be assimilated by mouth by lung or by rectum and cause spasm Some substances cause vascular lesions by pressure alone Novocain is an example of this latter group

*Lead* poisoning is partially a spastic vascular disease Some degree of vascular spasm is present early in all cases of lead poisoning Lead can cause a Raynaud's type of spasm The diagnosis is readily made if one thinks of the condition The history and examination with the finding of the lead line are significant The laboratory tests confirm the diagnosis

Nucleated red cells with anisopoikilocytosis stippled cells and an increase followed by a decrease in the blood platelets will substantiate the diagnosis The reticulocytes and basophils are also increased Lead may be found in the urine and feces

Bone changes due to lead absorption are a definite roentgenologic finding while the gastric upsets wrist drop and mental changes may or may not be present depending on the degree of poisoning

The treatment begins with the elimination of the causative metal The treatment is medical with a low calcium diet and 5 cc of phosphoric acid given every hour for twelve hours Epsom salts also is used routinely A positive calcium balance favors lead storage a low calcium and high phosphorus in a ratio of 1 to 3 is effective therapeutically in its elimination Surgically if gangrene develops amputation must be performed but this is a rare and late procedure

*Arsenic* has been described as causing Raynaud's syndrome

*Ergot* will cause a definite vasospasm [108] The spasm stages have been described as (*a*) cyanosis with recovery (*b*) deep cyanosis without blanching (*c*) necrosis It is necessary for the blood vessel constrictions to be maintained for many hours (forty-eight) for thrombosis to occur

The symptoms depend upon the degree of spasm and necrosis They may be vascular or neurologic or both In severe forms generalized symptoms with convulsions have been reported

Susceptibility to ergot must be present in the individual for the lesions to appear The widespread use of ergot in the postpartum period and in the treatment of migraine headaches without vascular disturbances indicates that an antithesis to the drug is necessary for vascular lesions to be produced

Treatment consists in elimination of the drug efforts to relax the spasm conservative management of any lesions produced and if gangrene is present amputation after demarcation is complete

*Adrenalin* and allied substances such as ephedrine benadryl etc may cause local necrosis by their vasoconstricting action Their effect is most often seen when they are combined in many stock preparations with novocain for local injections for anesthesia. The adrenalin causes the constriction of all blood vessels in that area While this reaction is of a transient nature at times it may cause permanent damage Consideration of the local effect of adrenalin should always be taken when this drug is used Where there are underlying vascular diseases its use is contraindicated It should not be used in the aged Widespread use of vasoconstricting substances for the common cold and sinus conditions may have a detrimental effect on patients with vascular diseases

*Novocain* itself by pressure may cause necrosis This is true of any solution if a sufficient quantity is injected to cause constriction of the blood vessels The "ring-like" type of block anesthesia used frequently for operations on fingers and toes may cause this necrosis In this type of anesthesia, a cuff-like infiltration is made at the base of the digit In order to be certain of the anesthesia, the quantity of novocain used or injected is often large, and pressure of the fluid at the point of injection closes the blood vessels and will cause the necrosis

A rubber band tourniquet on a digit increases the incidence of gangrene

*Hypodermoclysis* —In the days of hypodermoclysis in the lower extremities, necrosis occurred at times from pressure of the fluid in the vessels In such cases, there usually was some underlying vascular disease

*Infiltration* —The blood vessels will tolerate the injection of certain solutions which the subcutaneous and muscular tissues will not accept. No matter how perfect the intravenous technic, an occasional infiltration may occur In the patient with a vascular disease not only may there be a necrosis from infiltration but a spasm in the blood vessels supplying the area may develop This should be kept in mind and any such infiltration neutralized by dilution with procaine and saline and necessary therapeutic steps taken to counteract the spasm if such develops

## ACROCYANOSIS

Acrocyanosis is a spastic skin condition in which there is a duskiness and blueness of the extremity when exposed to cold There is usually edema and excessive sweating Acrocyanosis is frequently diagnosed as Raynaud's disease [71] The sex difference, the absence of regular attacks, and the rather characteristic lesions of acrocyanosis make it a syndrome in itself

**Etiology.**—Cold is a definite causative factor Patients with acrocyanosis have an underlying susceptibility to environmental changes, and a low temperature will precipitate the attack Wright[71] described many cases of acrocyanosis seen in patients who suffered from immersion foot and trench foot during World War II I saw many cases in Japanese prisoners from the Aleutian Islands

In contrast to Raynaud's disease where most of the patients are females, there is an equal distribution between both sexes in acrocyanosis Often the patients have an endocrine imbalance

The sympathetic innervation of the part is disturbed, as the symptoms of acrocyanosis are not unlike those resulting from overactivity of the sympathetic system

The lesion has been reported more often in patients with mental disorders than in normal individuals

**Symptoms.**—Cyanosis of the skin is not a constant finding, and there may be reddish to bluish areas, the color being deepest in the part which is more dependent Pressure will cause a paling and whiteness of the part, a point brought out by Crocq,[101] who was one of the first to describe the condition Coldness is a consistent symptom except in warm weather.

*Edema is a common symptom* and occurs in most of the patients with acrocyanosis

The sweating which occurs particularly on the soles of the feet or the palms of the hand is typical Heating of the part is not necessary for this symptom to develop This lesion may appear even if the patient is not irritated or emotionally upset

*Necrosis* —Ulceration and gangrene have not been reported and their presence would indicate that there is another disease complicating the acrocyanosis.

**Pathology** —Little is known of the pathology It is believed that the pathologic picture of acrocyanosis is that of an arteriolar obstruction This is in contrast to Raynaud's disease in which blood vessels larger than the arterioles are involved The arterioles apparently go into spasm Stern[107] has reported a hypertrophy of the medial layer of these vessels Edema with dilatation of the superficial capillaries is not unusual

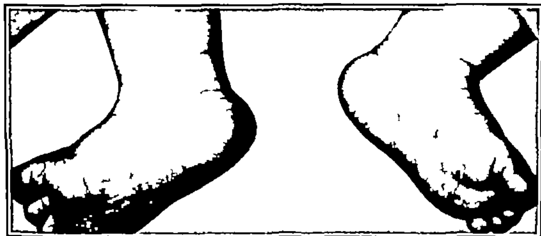


Fig 88.—Acrocyanosis in a one-year-old child present since birth No other symptoms except coldness.

The physiologic pathology is most likely an allergic manifestation of the small vessels to exposure to cold These vessels respond to a moderate degree of cold in a diffuse manner as brought out by Lewis[108] and Haxthausen.[109] A faulty vasomotor response in these vessels has been considered to be the pathologic physiology [71]

**Treatment.**—1 *Medical Treatment* —The medical treatment of acrocyanosis is mainly prophylactic The patient should be kept warm and protected from exposure to cold Any mental or psychic factor suspected as being the cause of the condition should be corrected The patient should be kept active as there is an increased incidence of the condition in those who remain in one position for a long time *i e* the trench foot or immersion foot patient

2 *Surgical Treatment* —A preganglionic sympathectomy as described on page 502 should be considered in patients with advanced acrocyanosis who do not respond well to medical measures Where the problem is mainly psychogenic in origin no relief can be expected from a sympathectomy

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Chapter

# 17

## ARTERIAL COLLATERAL CIRCULATION

THE body has the ability to supply collateral circulation around some divided or occluded vessels This is true particularly when the vessel is ligated and not thrombosed

When one or more of the major vessels are occluded, or traumatically or surgically divided, collateral circulation must be developed or death or loss of the part will follow This applies to occlusion of the following major arteries of the body

| | |
|---|---|
| Thoracic aorta | Abdominal aorta |
| Innominate artery | Common iliac artery |
| Subclavian artery | Hypogastric artery |
| Common carotid artery | External iliac artery |
| Axillary artery | Femoral artery |
| Brachial artery | Cerebral and coronary arteries |

Collateral channels are available when some of these arteries are occluded or divided

Some idea of the possible collateral branches after ligation of these arteries will be detailed A great deal of our information on collateral circulation was obtained from the study of anatomy books Unfortunately, the work on which such anatomical observations were detailed arose from autopsy specimens or the changes in an occasional patient These facts were obtained from patients who often had undiseased vascular systems It did not take into consideration the variations accompanying vascular diseases nor the fact that the entire picture changes depending upon whether the obstruction is an acute or slowly developing one

Aided by a financial grant by the United States Navy for the study of arterial occlusion, we were enabled to make studies on both animals in which occlusions were experimentally produced and on human patients who had occlusions due to disease The data now presented as to the development of collateral circulation are dependent upon that described in anatomical books but modified by our own findings in this study [5a] These changes vary with the age and physical status of the patient.

When there are arteriovenous fistulas present the danger of dividing the proximal artery increases, since that vessel carries the blood to both the periphery and the parasitic circulation which returns it through the fistula (See page 410 )

The importance of collateral circulation cannot be overemphasized Injury, disease or surgical operation may affect the circulation to any part

of the body While the arterial supply is the most important member of the circulation the ability of the venous and lymphatic channels to carry away the load must be acknowledged Failure on the part of either of these latter components will effect the primary one proportionately

Unfortunately Nature provided a larger number of alternative venous or lymphatic channels than she did in arranging collaterals to the arteries

The problem, as Learmonth[4] so well defined it is to maintain a sufficient flow of blood through the capillaries of the threatened part to suffice for its nutrition when it is at rest and to encourage the increase of this flow to suffice for its nutrition when it is physiologically active. This follows Poiseuille s law

$$\text{Quantity of blood varies with } \frac{(P_1 - P_2)r^4}{L}$$

$P_1$ = blood pressure at the arteriolar ends of the capillary loop
$P_2$ = blood pressure at the venous ends of the capillary loop
$r$ = the radius of the capillary
$L$ = length of the capillary

The radius of the capillary is most important as if this is only doubled the amount of blood flow is increased 16 times Since 94 per cent of the blood volume is either enroute to or away from the capillary bed and only 6 per cent is in the capillaries the main concern must be directed towards the blood flow from the heart to the capillaries. The blood volume must be maintained and the arterial pressure kept elevated without vasoconstriction This pressure is dependent upon the volume flow the internal resistance and the cross sectional area

Collateral circulation after obstruction to a major vessel may occur in one of two ways An alternative channel or channels may develop which continues the flow of the main artery If these collateral channels are short and the internal resistance is low the volume reaching the periphery may be equal nearly to the previous flow If the collateral channels are small or long the force of the flow may be depleted by the increased internal resistance In other instances of obstruction the arterial flow never returns to the main channels due to extensive arterial thrombosis. In such cases the part receives nutrition only from vessels of decreasing size and the blood reaching the capillary bed is much reduced in volume Prognosis in such cases is poor because the arterial pressure is reduced in overcoming the internal resistance of the smaller size blood vessels

**Pressure Fall From the Heart to the Periphery** —Determinations of the arterial pressure the internal resistance and the volume of flow are difficult to ascertain correctly due to the many factors inherent in the circulatory system such as spasm and vasomotor instability Investigators have made some determinations upon which an hypothesis can be based The systolic pressure in the aorta can be well determined and in the investigated animal the pressure in a terminal sized branch artery is available The outstanding work of Landis[4] in tabulating the average pressure in the arterial and venous ends of a capillary loop is of great value

Thus if our systolic blood pressure in the aorta is 120 mm of Hg the pressure when it reaches a terminal branch will be approximately 95 mm

of Hg When the flow reaches the capillary bed it will have fallen from 95 mm of Hg to 30 mm of Hg Thus, it takes 2½ times as much pressure to overcome the resistance in the arteriolar bed as it did to pass from the aorta through the large arteries, the main branches, and the terminal branches While Learmonth[6] showed that the total cross sectional area of the arterioles is only 25 times that of the terminal branches, the internal resistance in an arteriole is 1000 times greater than that in a terminal branch When the blood reaches the capillary bed, its flow is slow and the drop from the arterial end pressure of 30 to the venous end of 12 is only 18 mm of Hg In order to avoid ischemia after arterial occlusion, the main and terminal branches of the artery must be kept dilated by restorative or replacement methods and the arterioles also must dilate to increase the amount of blood reaching the capillaries[6] If the occlusion is slow, Nature can supply the alternative routes in most cases The patient of Chiene,[1] in which the celiac superior and inferior mesenteric arteries and the lower abdominal aorta had all suffered gradual obliteration with the supply of viscera and limbs maintained by somatic and subperitoneal arteries, is a case in point to illustrate the natural adjustment to gradual narrowing of the vessels

**Thoracic Aorta.**—The possibility of continued life after division of the thoracic aorta exists but will be dependent upon its gradual rather than sudden occlusion Cases have been reported and autopsies have shown coarctations of the aorta in which a collateral circulation existed Gray[3] reports two instances (Sydney Jones[9] and Wood[11]) The circulation was carried out by

(1) the internal mammary artery uniting with the intercostal arteries, with the inferior phrenic branch of the abdominal aorta by the musculophrenic and pericardiacophrenic branches and with the inferior epigastric artery,

(2) the costocervical trunk joining anteriorly with the first aortic intercostal artery and posteriorly with the posterior branch of the intercostal artery,

(3) the inferior thyroid artery developing a branch to join the first aortic intercostal artery

(4) the transverse cervical artery joining with the posterior branches of the intercostal arteries

(5) the subclavian and axillary arteries joined by means of enormous branches to the lateral branches of the intercostal arteries

Wood[11] found similar anastomosis in his case and noted that the blood supply of the abdomen and pelvis was mainly through the anastomosis of the intercostal arteries, while the supply to the lower extremities mainly was carried by the internal mammary and epigastric arteries

**Innominate Artery.** If the innominate artery is divided, the collateral circulation in the majority of patients could carry the circulation This would be carried on by branches from the carotid arteries of the opposite side, by the anastomosis of the costal cervical branches of the subclavian and first aortic intercostal into the right subclavian artery The numerous connections between the intercostal arteries and those of the axillary and internal mammary arteries also assist in getting blood to the arm The

inferior epigastric branch from the external iliac artery by anastomosis with the internal mammary would help in supplying the chest wall.

**Subclavian Artery.**—The subclavian artery because of its position at the junction of the neck and chest may be subject to injury either traumatically or surgically. If the subclavian artery is divided in its *third portion* the collateral circulation consists of three different vessels.

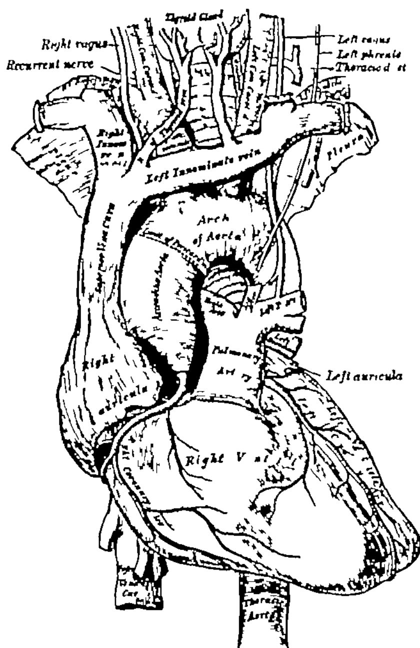



FIG 80.—The arch of the aorta, and its branches. (Gray's Anatomy.)

1. The transverse scapular and descending branch of the transverse cervical, both branches of the subclavian, anastomose with the subscapular from the axillary vessel thus carrying on the circulation.

2. The internal mammary vessels unite with the highest intercostal and lateral thoracic arteries and branches from the subscapular on the other side.

3 Small branches from the subclavian artery passing through the axilla terminate either in the main trunk or some branch of the axillary artery below the block These branches dilate immediately after ligation of the vessel.

Of these three paths, the subscapular artery, communicating freely with the internal mammary artery, transverse scapular, and the descending branch of the transverse cervical—all from the subclavian— is the most efficient This subscapular artery will dilate occasionally to 3 times the normal size.

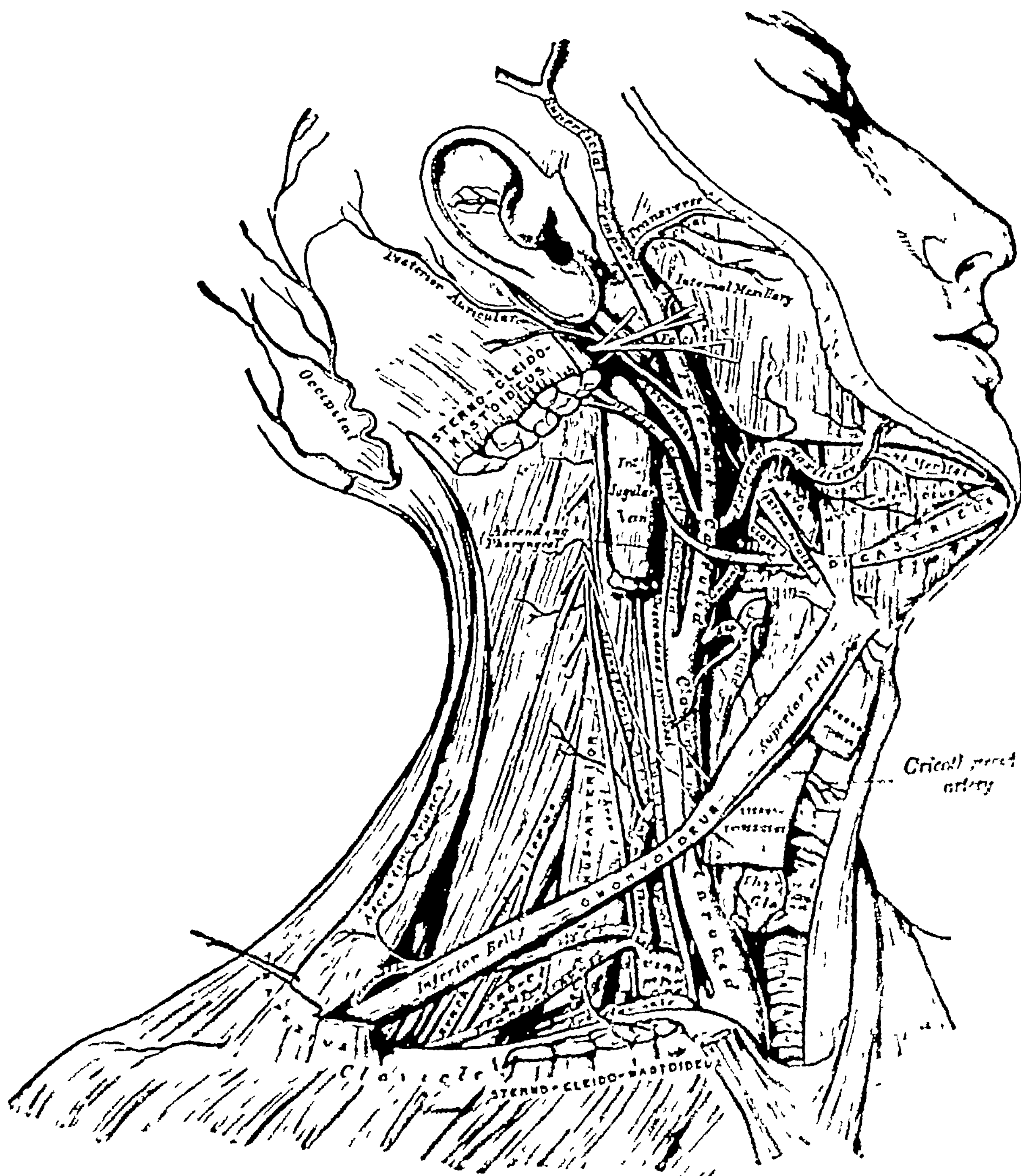



FIG. 90 Superficial dissection of the right side of the neck, showing the carotid and subclavian arteries (Gray's Anatomy)

en the subclavian artery is divided or obstructed in its *first portion* ation is carried on by an anastomosis of the superior and inferior id arteries and of the two vertebral arteries As a result, (1) the costal al branch anastomoses with the aortic intercostal arteries (2) the al artery anastomoses with the descending branch of the occipital , (3) the scapular branches of the thyroid-cervical trunk anastomose the branches of the axillary artery, and (4) the thoracic branches of xillary anastomose with the intercostal aortic arteries (see Fig 90) of interest that Halstead[1] was unable to find any evidence of gangrene ligation of the first part of the subclavian artery

**mmon Carotid Artery** —When the common carotid artery is divided unications take place from the other side between the superior and or thyroid arteries the deep cervical arteries and the descending ches of the occipital artery The vertebral artery takes over the func- of the internal carotid within the cranium There are also other ple communications The rich anastomosis between the two external id arteries helps return blood to the internal carotid arteries Sympa omy on the normal side aids such collateral circulation The incidence miplegia after ligation of the common carotid artery has been the ct of considerable controversy and is considered on pages 381 to 382

**llary Artery** —If the axillary artery is divided (1) above the *thoraco- ial branch* the collateral circulation will be the same as that in the part of the subclavian artery If the artery is tied below this point etween the *thoraco-acromial branch* and the *subscapular artery* the apular artery by its anastomosis with the transverse scapular and verse cervical branches becomes the chief collateral vessel (3) If the ry artery is divided lower the subscapular artery will again be the important point in the reestablishment of the circulation If the ion is *above* the *lateral thoracic branch* this vessel will aid by its junction the intercostal and internal mammary arteries. (4) If it is divided *the subscapular arteries*, the two humeral circumflex and subscapular nes by anastomosis with the deep brachial artery will help in circulation ration

**achial Artery** —(1) *Upper Arm* —Collateral circulation will be from numeral circumflex and subscapular arteries to the ascending branches e deep brachial artery

) *Lower Arm* —Below the brachial profunda artery and the superior r collateral the circulation is carried by these arteries branches ng with the inferior ulnar collateral and the radial and ulnar recurrent ies as well as the dorsal interosseus arteries

**bdominal Aorta.**—If the abdominal aorta is divided (1) between the rior mesenteric and inferior mesenteric vessels the collateral circulation ependent on anastomosis between the internal mammary and the ior epigastric arteries and by anastomosis between the superior and ior mesenteric vessels Such collateral circulation rarely is sufficient ) If the division occurred *below* the inferior mesentery (usual) then tomosis between the inferior mesenteric and the internal pudendal ries is the greatest source of collateral circulation, in addition the lum arteries anastomose with branches of the hypogastric artery Patients e survived slow occlusion in this area

**Common Iliac Artery.**—When the common iliac artery is divided or ligated, the collateral circulation is provided in the following ways:

(1) The hemorrhoidal branches of the hypogastric artery anastomose with the superior hemorrhoidal branch from the inferior mesenteric artery.

(2) In the female, the uterine, the ovarian, and the vesical arteries of the side opposite to the divided common iliac artery anastomose with those on the side of the divided artery.

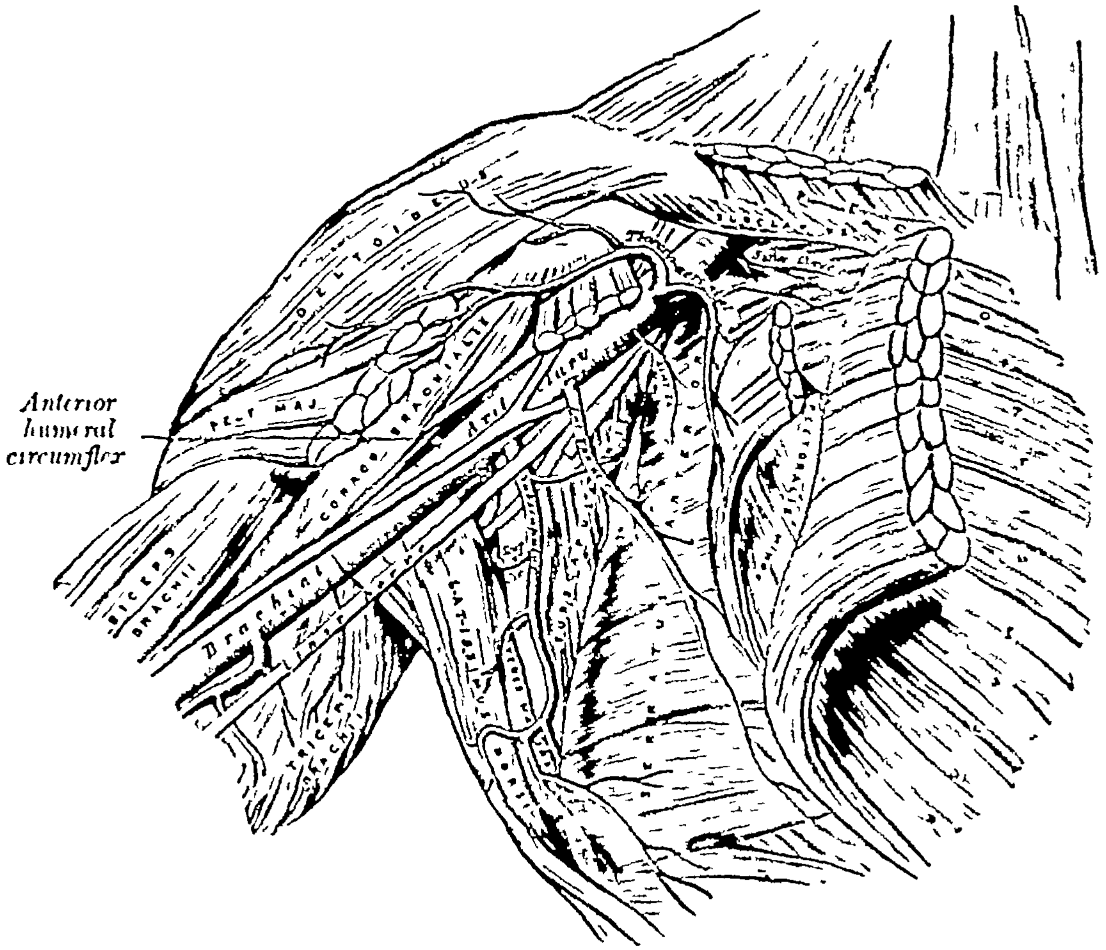



FIG. 91.—The axillary artery and its branches. (Gray's Anatomy.)

(3) The lateral sacral and the middle sacral arteries supply collateral circulation to the opposite side.

(4) The inferior intercostal and the lumbar arteries also take over some of the burden of circulation.

(5) The inferior epigastric artery anastomoses with the internal mammary arteries.

(6) The deep iliac circumflex artery anastomoses with the lumbar arteries.

(7) The iliolumbar artery anastomoses with the lowest lumbar artery.

(8) The obturator artery by its pubic branch may anastomose with the obturator artery on the opposite side and with the inferior epigastric artery.

**Hypogastric Artery** —If the hypogastric artery is divided or ligated the collateral circulation is carried on by anastomosis of the following vessels

(1) The two uterine arteries in the female

(2) The two ovarian arteries in the female

(3) The vesical arteries on each side

(4) The hemorrhoidal branches of the hypogastric artery with the hemorrhoidal branches of the inferior mesenteric artery

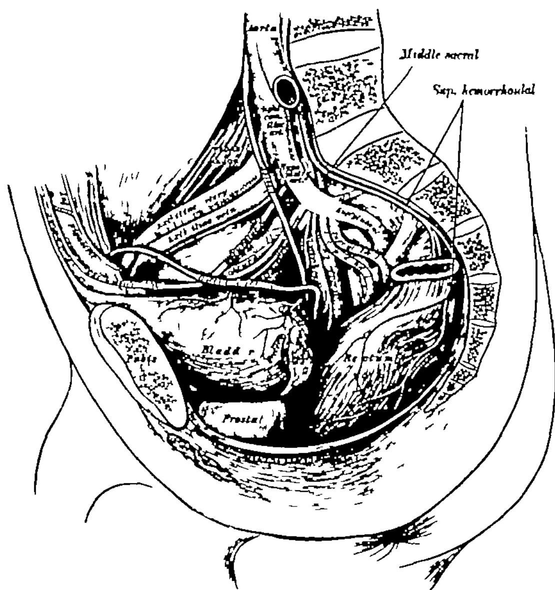



Fig 92 —The arteries of the pelvis. (Gray's Anatomy )

(5) The pubic branch of the obturator artery with its opposite vessel and with the inferior epigastric and medial femoral circumflex artery

(6) The perforating and circumflex branches of the profunda femoral artery with the inferior gluteal artery

(7) The superior gluteal artery with the posterior branches of the lateral sacral arteries.

of choice Where this is not possible or feasible, efforts must be directed to the following

(*a*) Development and dilatation of the collateral circulation to its greatest potential by maintaining the volume and the arterial pressure

(*b*) Avoidance of any physical or chemical constriction

(*c*) The addition of anticoagulant factors to the blood to prevent and minimize the thrombosis inherent in the occlusion and the slowed blood stream

(*d*) Restriction of the demands of the part to those that can be supplied by the reduced blood supply

(*e*) Time is an important developer of collateral circulation Definitive surgery should be delayed as long as possible dependent upon the individual problem This is true particularly in the operative eradication of aneurysms

(*f*) Permanent interruption of the sympathetic nervous system to the affected part

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Chapter
# 18

## TRAUMATIC INJURIES TO ARTERIES

### *War Wounds Industrial Injuries Accidents*

THE injury to a major artery is a catastrophe which will be encountered more frequently in this age of rapid and distant transportation It has been a problem in war and traumatic surgery since the days of the cavemen Survival of the affected part and the individual depends upon the management of the injury Injuries to arteries can be divided logically into those in which the continuity of the vessel injured is necessary for life or viability of the part and those in which collateral circulation is adequate as to require only control of the local lesion Examples of the first type are the aorta the common iliac artery and most of the end arteries to the mesentery to the heart to the kidney liver brain and the end arteries such as the popliteal Examples of the second group are most superficial lacerations and those around the neck and face

The fact that injured major arteries can be repaired was not accepted for many years and even today many surgeons do not recognize the efficacy of suturing or replacing injured arteries

Many times even a slight injury to an artery is treated by ligation and division of the vessel In the hands of the unskilled surgeon such a ligation may be lifesaving It has been demonstrated so frequently, however that an artery can be successfully sutured or replaced that this arterial suture technic should be acquired and practiced by all surgeons (See pages 335 to 330 for details)

Laceration of major vessels may occur during an operation as well as following trauma The ligation of a major artery if not followed by gangrene will result in changes in the vascular supply to that part usually of a permanent nature, and the patient may be made a vascular cripple needlessly This unnecessary ligation of arteries should be eliminated

When such ligations are required the fate of the part depends upon the *collateral circulation* available and the *site of the ligation* The former point the collateral circulation has been discussed in the previous chapter The reader is directed to this chapter and particularly to the pages where Poiseuille s law and the fall in pressure gradient are discussed (see pages 310 to 320) It is on the conservation and correct use of this collateral circulation that the ultimate result will depend

The site of ligation also is important Gangrene follows the ligation of some arteries at certain points almost invariably For example the ligation of the superficial femoral or brachial arteries will cause gangrene and necessitate amputation more frequently than the ligation of other arteries

In these two arteries there are blind segments distal to their main collateral branch in which the collateral branches are absent or minimal. If the artery is ligated some distance distal to the main collateral branch, a blind segment of artery or arterial pouch remains, into which the arterial blood is directed with dissipation of the arterial pressure in distension of this pouch. This detracts from the arterial pressure of the blood entering the main collateral branch of the artery.[34]

If the superficial femoral artery is ligated immediately distal to the femoral profunda, gangrene is extremely rare. If a vessel must be ligated, it should be tied if at all possible just distal to a large collateral branch so that the entire force in the vessel at the point of ligation will be directed toward this collateral vessel.

TABLE 23.—INCIDENCE OF GANGRENE IN MAIN VESSEL INTERRUPTION

| *Artery* | *Ligation Only* | *World War I* Wound and Ligation* | *World War II** Wound and Ligation* |
|---|---|---|---|
| Subclavian | 0 | 8.8% | 24.0% |
| Axillary | 1.4% | 2.7% | 25.0% |
| Brachial | 0 | 4.0% | ? |
| Femoral | 17.2% | 20.2% | 32.0% |
| Popliteal | 26.6% | 34.7% | 86.0% |

* Figures for World War I are according to Makins, as quoted by Luke.[46]

** Figures for World War II are approximate and are based on reports from various sources.

Muscles around such a vessel take the force of the pulsation coming down the vessel to the ligated point. They absorb the pulsation so that it does not continue along the small collateral vessels to enlarge them, as is necessary if the part is to survive.

Thus both the collateral circulation and the site of ligation are important and determine the life or death of the part after an artery is ligated.

While the gangrene incidence varies with the vessel involved and other factors, the figures shown in Table 23 indicate that if untreated, a high percentage of those suffering main vessel interruption will develop gangrene.

Statistics on the incidence of gangrene in the American Third Army during the war on the European continent, from August 1944 to May 1945, showed that of 837 major blood vessel injuries encountered, 423 or 50 per cent required a major amputation.

This amputation rate was reduced to 38 per cent in those in whom vessel suture or vein grafting was carried out.[46] See page 334 for details on vascular sutures and page 339 for discussion on vein grafting. This figure still is a high one. It has greatly been reduced in the Korean war (see page 327).

**Symptoms.**—The symptoms of an injury to an artery vary with the site and the degree of the laceration and, secondarily, with the patient's response to the trauma. Other factors enter the picture such as the age, the injuring agent and the healthy or diseased status of the blood vessel. If an artery is lacerated, in addition to the signs at the injury site, there usually will be signs of vasoconstriction and spasm in the affected part distal to the injury

When a major undiseased artery is injured spasm results—the so-called steuper arteriale described by the French While intrinsic contraction of the smooth muscle in the vessel itself may play some part it is more likely due to sympathetic vasoconstriction This theory is proven by the

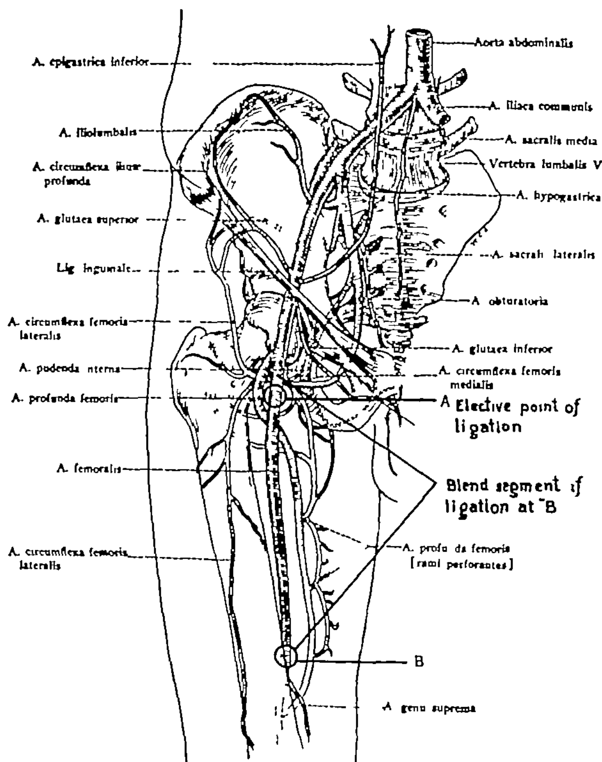



FIG 94 —Anatomy of the femoral artery (Eycleshymer and Jones.) If it is necessary to ligate such vessels it is best to ligate just distal to the profunda, at A rather than a lower point, B This directs the force of blood into the profunda artery rather than into a blind end of the femoral artery

relaxation of the vessel which occurs under sympathetic nerve blocks This spasm is physiologic and may be lifesaving at the time, as it reduces hemorrhage and squeezes out blood which might clot in it and in the collateral vessels Spasm should not be relaxed until hemorrhage control or arterial repair or replacement, if possible, has been achieved

The limb becomes white and the pulse may be absent or restricted The oscillometric readings likewise will be absent or markedly reduced The foot may become somewhat warmer due to venous congestion and with the continuation of spasm it gradually cools Mottling from the venous

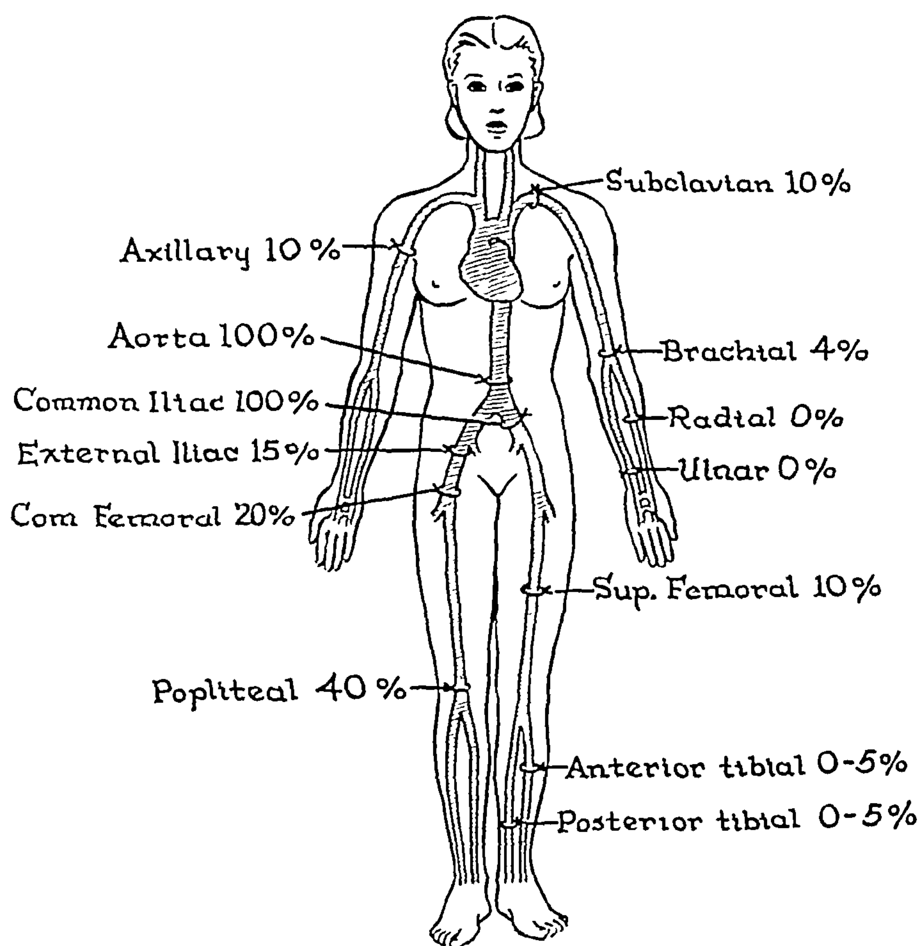



Fig 95 —Expectation of gangrene by ligation at various points in the arterial tree (Based on multiple figures )

congestion follows The limb may or may not be paralyzed, depending upon whether the blood supply to the muscles or nerves which move the part has been shut off or not

At the site of injury there will be hemorrhage and, at times, actual arterial spurting of blood unless controlled by tourniquet or by spasm A large tumor mass, due to a blood clot, may be present and this clot temporarily may have occluded the vessel If the veins are also lacerated, arterial blood may be pumped into the venous tree with dilatation of the veins, but this occurs later

**Differential Diagnosis**.—Where there are symptoms and evidence of a direct trauma to an artery the following diseases may have to be differentiated

(1) *Arterial Thrombosis*.—The history of the type of trauma is a significant point Previous arterial disease makes the vessel more prone to thrombosis spontaneously or on a traumatic basis

(2) *Arteriosclerosis*.—The loosening of an arteriosclerotic plaque may simulate a laceration of a major artery Again the history calcification elsewhere and the roentgen ray findings help make the diagnosis

(3) *Reflex Vasospasm*.—This may give the appearance of arterial failure This should not be difficult to differentiate if the possibility is considered A sympathetic block will help in the diagnosis

**Treatment.**—THE EMERGENCY TREATMENT OF ARTERIAL INJURIES.—A *The Control of Hemorrhage*.—The first and paramount point in the treatment of injury to a major vessel is the control of hemorrhage This seems obvious but requires emphasis For example patients have reached first aid stations or hospitals nearly exsanguinated when mere pressure could have controlled blood loss Nature has provided us with the perfect hemostat—the fingers The panic of the attendant that follows blood loss must be controlled While replacing fluids combating shock raising blood pressure and reducing the pulse rate are all necessary one must close "the hole in the dyke" first There is no reason for raising the blood pressure and increasing the cardiac output first because this overcomes Nature's efforts to control the hemorrhage The blood pressure is down and the patient is in shock not only because of blood loss but also because the shock is a physiologic response defensively to reduce the blood loss Such simple measures as pressure digital compression elevation and sedation are advocated to replace the previous first aid therapy of a tourniquet

In war time it is important that not only the corpsmen but all men in combat have some knowledge of hemorrhage control The battalion surgeon should explain to the men the simple anatomic and physiologic problems which arise in hemorrhage *Arterial bleeding* from a major vessel can exsanguinate an individual in a very short time Paradoxically this bleeding often is the simplest to control Continued *venous bleeding* is not as apparent but may be more deadly in its results It is always controllable by pressure.

(1) *The Tourniquet Problem*.—From the earliest times the tourniquet has been advocated for the emergency control of hemorrhage Lives have been saved undoubtedly by this method and in untrained hands the use of a tourniquet has a place in therapy The part becomes ischemic when a tourniquet is applied and the danger from application is great First aid attendants should be trained to try first to control hemorrhage without the use of a tourniquet If a tourniquet is applied effectively the limb becomes ischemic. The time that a limb can remain in such a state without irreparable damage is limited Thus the *tourniquet* should be *reserved* for only such bleeding as is uncontrollable by all other means

The first aid attendant must know however that if other measures are not effective promptly the tourniquet should be applied The responsibility and the patient's priority for prompt evacuation increases with the

application of a tourniquet. If used, it should be applied, proximally, as closely to the wound as possible. Any piece of gauze, cloth or leather belt will be effective and the addition of a windlass increases the power of its application. A tag with the hour and the minute of its application should be attached. If evacuation has to be delayed, the tourniquet should NOT be released momentarily every one or two hours, as has been advised repeatedly. This leads to increased blood loss and even exsanguination. Army and Navy directions in this respect have been revised.

(2) *Pressure.*—Pressure will control most active arterial bleeding in the acute emergency. The number of patients alive today because an individual knew enough to put his finger against or above a wound from which blood was spurting is legion. *Venous bleeding*, likewise, may be exsanguinating. It is a slower but more insidious blood loss, but its less demonstrative nature may make it more malignant. Such bleeding from an extremity can always be controlled by pressure aided by elevation.

(3) *Hemostat Control.*—The ideal control of bleeding vessels is by hemostat with or without ligature. This type of therapy is limited to doctors or experienced attendants. Hemostatic control of venous bleeding with ligation of the vessels where possible is preferable to other methods of control.

(4) *Packs.*—A sterile gauze pack will control venous and most small arterial bleeding. This pack may be augmented by pressure and elevation of the part. This type of hemorrhage control has obvious advantages over the application of a tourniquet. The patient should be tagged with the hour and minute of the pack application and a description of the wound made to include an estimate of the amount of blood loss. The status of the circulation distal to the pack should be observed regularly thereafter.

(5) *Protection of the Collateral Circulation.*—The injury to a major vessel may mean the permanent loss of that blood supply. The circulation to that part then will depend upon the small branches of the main artery, or the collateral vessels. These vessels should be preserved. If immediate clamping or suturing is performed, only the injured vessels should be ligated. If the wound must be enlarged to control the bleeding, as few collateral vessels as possible should be divided.

(6) *Vasospasm.*—Spasm plays a major part in the pathology of wounds of major vessels. Its therapy will be discussed on pages 344 and 345. At the time of the original injury, spasm aids in the control of hemorrhage. After the bleeding is controlled, as part of the first aid the spasm should be relieved.

(7) *Foreign Hemostatic Agents.*—Certain substances such as gelatin sponges and fibrin foams have hemostatic properties. The hemorrhage from the wounds of the coronary vessels has been stopped completely in experimental animals without the use of a ligature by applying gelatin sponge.[38] These sponges have been applied by a "patch" method on wounds of the auricle, ventricle, and liver. These and other hemostatic substances will have an increasing part to play in the control of hemorrhage. Experience is required for their successful application, but the inclusion of these hemostatic agents in the first aid equipment in the future will be of value.

(8) *The Effect of Tobacco* —No injured patient should be allowed to smoke. The effect of nicotine as a cause of vasoconstriction has been proven time and again.[10,14,41] Experimentally one can observe small vessels in the rabbit's ear blanch out with the injection of minimal amounts of nicotine. As many limbs have been lost from continued smoking in patients who have arterial occlusive disease as in those who have been injured. *The importance* of the spasm of any injured vessel which follows smoking *cannot be overemphasized*. The vasoconstriction caused by smoking in the patient with a traumatic wound may cause even greater damage to the remaining vessels than we know it does in those with an arterial disease because sufficient time has not elapsed to permit collateral vessels to develop.

TABLE 24 —RESULTS OF ARTERY REPAIR IN THE KOREAN WAR

| | | | *Treatment* | | | *Loss of Limb* | |
|---|---|---|---|---|---|---|---|
| *Vessel* | *Total Cases* | *Per Cent Total* | *Anast* | *Vein Graft* | *Trans Repair* | *No Cases* | *Per Cent* |
| Carotid | 1 | 3 | 1 | | | | |
| Axillary | 5 | 15.2 | 4 | 1 | | | |
| Brachial | 10 | 30.4 | 9 | 1 | | | |
| Above Profunda | 4 | 12.1 | 4 | | | | |
| Below Profunda | 6 | 18.1 | 5 | 1 | | | |
| External Iliac | 1 | 3 | 1 | | | | |
| Femoral | 9 | 27.2 | 6 | 2 | 1 | 1 | 11.1 |
| Above Profunda | 1 | 3 | 1 | | | | |
| Below Profunda | 8 | 24.3 | 5 | 2 | 1 | 1 | 12.5 |
| Popliteal | 7 | 21.2 | 6 | | 1 | 2 | 28.5 |
| Total | 33 | 100 | 27 | 4 | 2 | 3 | 9.1 |

Above figures courtesy of Jahnke, Edw. J., Major, USAF (MC), Walter Reed Hospital. (Published Ann. Surg. *138*: 168, 1953.) Figures from Army Research Team in Korea.

(9) *Care of the Patient as to Warmth and Further Trauma* —The patient should be kept warm and further injury to the parts should be eliminated. Many patients are traumatized during evacuation or movement. No patient with a vascular wound of his lower extremities should walk from the site of injury unless there is further danger to his life without evacuation (*i.e.* war wounds).

B. *Resuscitation of the Injured Patient* —With control of the hemorrhage the resuscitation of the patient is next in importance. Fluid and blood loss must be replaced as the reduction in the blood volume will reduce the amount of blood flow through the peripheral vessels. This loss further impairs the circulation distal to the injury. In war or in a major disaster in the first aid area, this may require the use of some blood substitute such as plasma or gelatin. Whole blood loss should be replaced as rapidly as possible because sufficient oxygen carriers to replace the lost red blood cells are necessary. Fluid and blood replacement must be continued during and after evacuation until normal levels of hemoglobin, serum

blood volume and blood cells have been restored The plasma expanders may be the answer to this problem in major disasters or future wars

*The Relief of Pain* —The relief of pain by the use of sedatives is of primary importance Pain in itself produces shock Sedatives and opiates, however, should not be administered to the unconscious patient

TABLE 25 —DISTRIBUTION OF 77 CONSECUTIVE VASCULAR INJURIES TREATED AND INCIDENCE OF GANGRENE

| | | | *Loss of Limb* | |
|---|---|---|---|---|
| *Vessel* | *Total Number* | *Per Cent Total* | *No Cases* | *Per Cent* |
| Common Carotid | 1 | 1 3 | | |
| External Carotid | 1 | 1 3 | | |
| Axillary | 5 | 6 6 | | |
| Brachial | 12 | 15 7 | | |
| Above Profunda | 4 | 5 2 | | |
| Below Profunda | 7 | 9 0 | | |
| Profunda | 1 | 1 3 | | |
| Radial | 6 | 7 9 | | |
| Ulnar | 5 | 6 6 | | |
| Radial and Ulnar | 2 | 2 6 | | |
| External Iliac | 1 | 1 3 | | |
| Femoral | 14 | 18 5 | 2 | 14 2 |
| Above Profunda | 1 | 1 3 | | |
| Below Profunda | 8 | 10 6 | 1 | 12 5 |
| Profunda | 5 | 6 6 | 1 | 20 |
| Popliteal | 7 | 9 0 | 2 | 28 5 |
| Anterior Tibial | 6 | 7 9 | 1 | 16 6 |
| Posterior Tibial | 9 | 11 4 | 3 | 33 3 |
| Both Tibials | 2 | 2 6 | | |
| Peroneal | 3 | 4 0 | | |
| Anterior Tibial and Peroneal | 1 | 1 3 | | |
| Posterior Tibial and Peroneal | 1 | 1 3 | | |
| Both Tibials and Peroneal | 1 | 1 3 | | |
| Total | 77 | 100 | 8 | 10 6 |

Above figures courtesy of Jahnke, Edw J, Major, USAF (MC), Walter Reed Hospital (Published, Ann Surg, *138*, 161, 1953) Figures from Army Research Team in Korea

After the blood loss is replaced, and pain is relieved, the patient may need other supportive measures in his resuscitation *Oxygen* may be needed, but this usually requires evacuation from the site of injury Resuscitative drugs such as caffeine and adrenalin compounds should be given with due regard to the possible vasoconstrictive effect of such drugs The relief of pain, warmth and posture changes all are important in the relief of shock

C *General Therapy* —General measures include the therapy of shock, immobilization with adequate splinting of the injured part, control of pain and the treatment of associated wounds Antitetanic and antibiotic therapy must be started at once

D *Evacuation* —Vascular injuries pose special problems in the movement of the patient

During warfare front-line gunshot bayonet mortar or shrapnel wounds and the locality of the fighting make first aid care difficult Not only must the medical corpsmen or the wounded man s companions attempt to extricate him from his precarious position without further injury but they *must do so while protecting their own lives.* In such efforts more damage may be inflicted to the wounded man In a major military advance a short delay to secure the area around the wounded man before evacuation will increase the survival rate in those injured If hemorrhage can be controlled evacuation may be delayed until transportation is available The same applies to major peacetime disasters Adequate splinting is of primary importance

TABLE 20 —RESULTS OF ARTERIAL REPAIR

| Artery | Number Cases | Type of Repair: Anast | Graft (Vein) | Transverse Suture | Result No Amputations |
|---|---|---|---|---|---|
| Common Carotid | 3 | 2 | 1 | 0 | 0* |
| Subclavian | 1 | 0 | 1 | 0 | 0 |
| Axillary | 7 | 5 | 2 | 0 | 0 |
| Brachial | 16 | 15 | 1 | 0 | 0 |
| Ext Iliac | 2 | 2 | 0 | 0 | 1 |
| Com. Femoral | 2 | 2 | 0 | 0 | 0 |
| Sup Femoral | 13 | 10 | 3 | 0 | 2 |
| Popliteal | 14 | 12 | 0 | 2 | 3 |
| Total | 58 | 48 | 8 | 2 | 6 |

* No neurological sequellae

Figures from Army Research Team in Korea.

(1) *Movement of Patient* —The general treatment of all wounds especially applies to injuries involving the vascular system An insecurely held fractured bone may cause permanent vascular trauma during movement Foreign bodies splinters or missiles may cause irreparable damage to fragile blood vessels It is so important to immobilize the part that this admonition is repeated to prevent further or new vascular injuries The bandage or cast must be applied so as not to cause pressure on the blood vessels If the patient complains of coldness or numbness or if there is any change in color or temperature the bandage or wound must be inspected and readjusted if needed The fragile blood vessel must be kept in mind as the patient is transported from the first aid attendant through the various individuals care until he reaches the final specialist who will perform the definitive surgery The danger of injuring the blood vessel can be as great in moving a patient from the stretcher to the operating table in the hospital as it can be in dragging the same individual over a rough terrain in warfare into a foxhole.' Splints casts and the pinning of fractures with the incorporation of these pins into casts to prevent the

rough bone ends from moving during evacuation movements in World War II were responsible for the salvage of innumerable limbs

OPERATIVE TREATMENT — A *Decision as to Operation* — With the hemorrhage controlled and the patient resuscitated, a short period of careful observation is indicated The status of the remaining circulation can be ascertained The relief of the spasm and recovery from shock often allows the remaining circulation to take over for the damaged major vessel

*Exploration is indicated only where there is continuing hemorrhage or there is evidence of impending gangrene distal to the injury* It may be elected to prevent secondary hemorrhage at the ideal operative time when sufficient blood replacement and skilled assistants are available Sympathetic nerve blocks, intravenous procaine, and slight dependency of the part may aid the restoration of the blood supply if the distal circulation is failing If there is no improvement within ten to twelve hours an exploratory operation must be made If the six "P's" of ischemia are present operation always is indicated During this observation time, venous engorgement by back pressure in the capillary bed, or subcutaneous bleeding, may change the pale white color to a dusky redness

SIX "P's" OF ISCHEMIA

| | |
|---|---|
| (1) Paralysis | (4) Paresthesia |
| (2) Pulselessness | (5) Paleness |
| (3) Pain | (6) Prostration |

The operation should be performed at the first hospital or, in war times, at the evacuation site at which the proper facilities, both as to equipment and personnel, exist With rapid air and ship evacuation, definitive blood vessel surgery within the optimal twelve hours after injury will be feasible more often in future conflicts than in the past

B *Preparation to Replace Blood Loss* — Wounds of the vascular system are potentially dangerous at operation time One must be prepared for severe hemorrhage. The bleeding may have been controlled temporarily, but operation often removes this control Blood must be available in adequate quantities Both intravenous and intra-arterial blood replacement may be required For major vessel injuries 3000 to 5000 cc should be available *in the operating room* A cut-down, with a cannula or tube tied into the vessel, is advised The surgeon can physically control the bleeding area with his hands or fingers should severe or serious hemorrhage occur, until the initial loss has been compensated Trained assistants, correct instruments and suction reduce the blood loss danger

C *Types of Injuries to the Artery* — The injury to the artery may be indirect, such as a contusion, or a direct injury The indirect injury results in a contusion to the artery without necessarily a laceration of the wall The direct lacerations may be partial, a perforation or a *complete severance* of the vessel wall In the latter type the division may be accompanied by the loss of part of the vessel In the *partial laceration* there is an incomplete tear through the artery wall In such a laceration, part of the vessel remains in continuity while the other part is cut or torn

A *perforation* of an artery most often is caused by some small missile or a stab, but it can be caused by flying debris also, particularly glass

In the third or complete type of laceration there is an actual division of the vessel This can be caused by a stab or bayonet wound or by a small missile or flying debris

The fourth type of laceration is that in which there is complete division of the vessel *with the loss of a segment* of this artery Usually such an injury is accompanied by marked loss of the soft tissues and bone Major accidents in cars trains or airplanes or industrial plants are common causes for such an injury In war times shrapnel mortar fire rockets and bombs are the usual cause of such an injury This loss of continuity may be small or may involve most of a large vessel The part usually will be lost in such an injury as it is usually impossible to approximate the ends of these divided vessels The collateral circulation generally is destroyed An analogous vein or homologous artery graft may be tried to restore the circulation to the part This type of procedure is not a first aid or evacuation operation but may be done at a definitive surgical site

D *Exploration of Injury to Artery* —The usual preparation of the skin is made The incision should begin above the injury site in the anatomical direction of the major artery involved This incision permits control of the vessels both proximal and distal to the wound Such control should be secured before disturbing the wound *per se* and the incision should be extended as far as is necessary for control Any bone obstruction which might interfere with adequate blood vessel control must be removed In vessels arising from the aorta the ribs clavicle or sternum should be removed if necessary as the patient may be lost from hemorrhage because the undamaged proximal vessel cannot be reached rapidly enough in an emergency *Distal* as well as *proximal* control of the injured vessel is required as serious bleeding may occur from the distal end of an artery due to collateral supply even though the proximal end has been occluded The ischemia and spasm of the vessel may cause the pulsation on which one relies anatomically to be absent The identification of vessels nerves and tendons as such in severe wounds may depend upon their anatomical position An anatomy book should be available in the operating room and consulted Rubber bands held loosely by hemostats should be placed around the artery and vein for control of later hemorrhage The incision then should be continued directly to the site of the trauma with excision of the wound of entrance Dead and necrotic tissue or foreign bodies are cut away or removed

(1) *Contusion of an Artery with Thrombosis* —The artery may be thrombosed without an active laceration of the vessel The vessel may have been crushed or torn secondarily to injury of the soft tissues In a contusion this site will be shown by subcutaneous bruising and hemorrhage If the thrombus is palpable it may be removed through a small incision in the artery Secondary clots both distally and proximally should be teased out with a smooth forceps and suction This maneuver is most important and may require ingenious methods (see *Thrombectomy* Chapter 14) The vessel then should be irrigated with a solution of saline and heparin Heparin solution should be left in the lumen and the artery incision closed with fine arterial silk If thrombectomy fails and an adequate blood flow cannot be obtained one may consider a graft If this is impossible it is

safer to divide and ligate the vessel The ligation should be performed at an uninjured site See page 333 for technic and sites of election for ligation If ligation is necessary, subsequent success or failure of the circulation depends upon the collateral circulation and supply, and requires the relaxation of the reflex arteriospasm.

(2) *Laceration of an Artery*—With the proximal and distal control secured, the point of missile entry in a *laceration* is excised with the surrounding skin, foreign bodies and devitalized tissue The hematoma then is evacuated The proximal control alone will not be sufficient to stop the bleeding and this point is repeated for emphasis Collateral circulation from above the rubber-taped area may bring large amounts of blood into the wound through the distal end of the vessel Serious active bleeding may occur from the distal end of the *vein* if it has been severed The extent of the injury is determined, as to the complete or partial division of the artery and vein and whether a segment of the vessel is lost The decision as to what procedure to perform then must be made This decision requires general surgical judgment and experience in addition to the technical ability to suture a blood vessel If trained personnel are not available, conservative ligation of the vessel is the safest procedure The wound should be closed loosely or not at all

(3) *Incomplete Laceration of an Artery*—All such wounds should be treated, like other traumatic wounds, with surgical cleansing and debridement of dead tissue or foreign bodies After control of the vessels is obtained, in an *incomplete laceration* the accompanying thrombus may be removed If the wound is not grossly infected, the edges may be freshened and sutured using fine arterial silk (see *Technic of Arterial Suture*, p 335) Where the collateral circulation is adequate, or where the status of the artery wound makes a repair not advisable, the artery should be divided and sutured or ligated Injured veins should be opened, aspirated proximally for clots until a free flow is obtained, and ligated

*Ligation of an Artery*—The principles discussed under exploration should be followed (see pages 330 to 331) The ligation of a major artery does not necessarily mean the death of the part or tissue distal to the ligation In emergency or front-line surgery, or where there are not adequate facilities, ligation of a lacerated artery often will be the treatment of choice. If the time interval since the injury is protracted, and the leg obviously ischemic, it is the elected treatment If the ligation is performed at a bruised, infected or devitalized point, it may slough and give way to secondary hemorrhage A ligation at a healthy area reduces the danger of a thrombus extension into collateral vessels and removes that injured vessel site as an originator of vasoconstriction The vessel should not be ligated without division The vessel may reopen or slough with a secondary hemorrhage and this is not good surgical practice

(1) *Point of Election of Artery Ligation*—The point of election for ligation of an artery is just distal to a major branch Ligation at a distance from a main branch creates a blind pouch into which the thrust of the arterial flow is directed and lost This detracts from the force into other collateral vessels[31,60] In an elective procedure, this point is worthy of note In traumatic wounds, however, where dissection and opening of new tissue

spaces is necessary to reach such a ligation point, it is not practical nor indicated The possible spread of infection further contraindicates such dissection

(2) *Technic of Ligation of a Major Vessel* —The vessels should be dissected to a viable and uninjured area Clotted and fluid blood should be removed from the vessel at the point of ligation thus one ligates a flaccid vessel rather than a full tense one[31] A free ligature is then applied to the artery The ligature is placed sufficiently proximal to the end of the artery so a transfixion suture can be inserted distally[22] A transfixion suture on a fine atraumatic needle is inserted and ligated The suture material of choice for these vessels is silk The size of this *ligature* need be only as large as permits its tying without breaking The silk should not have

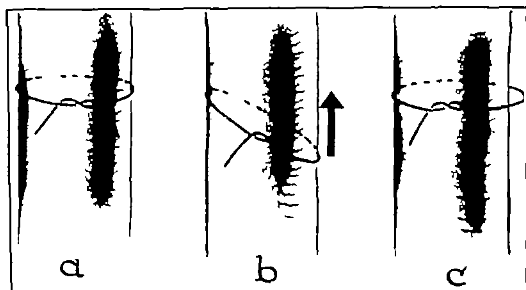



Fig 96 —Ligation of an artery Technic of ligating a major vessel The sutures must be at right angles to the vessel thus a round and not an oval type. The knots must be square and "lie down"

From author's work published in Surgery of Trauma Edited by Bowers, W F Colonel M C U.S.A., Chapter 15 Regional Wound Surgery Section 2 Vascular Wounds p 268 J B Lippincott Company Philadelphia, 1953

been over-boiled or sterilized too often and should be tested for tensile strength For vessels the size of a femoral artery 00 or 000 silk is a satisfactory size[24]

*Tying the Suture on a Major Vessel* —The knots must be square and they must lie down correctly The "crossed hand technic assures this point Young surgeons must learn this technic. If the knots are not flat they may loosen with the movement of the part The ligature must encircle the artery at right angles to it at all points The ligature thus must be of the *round* and not *oval* type If the vessel is ligated at different levels on the two sides the ligature later may slide until the two sides are equal Since an oval is larger than a circular tie the tie will be loose on the vessel It is unnecessary and unwise to clamp the vessel at the ligation point A clamp bruises the walls of the vessel particularly the endothelium

and may initiate a propagating thrombus Bleeding can be controlled by pressure or a rubber band or tape If the vessel is clamped, the suture should be placed proximal to this traumatized area

*Suture Versus Ligation of an Artery* —To save vessel length, preserve collateral branches and avoid slipping, the suture technic is more satisfactory than ligation A fine suture can be used During the suturing, a rubber band temporarily occludes the vessel The end is sutured with an atraumatic 0000 (4-0) or 00000 (5-0) silk The incidence of hemorrhage

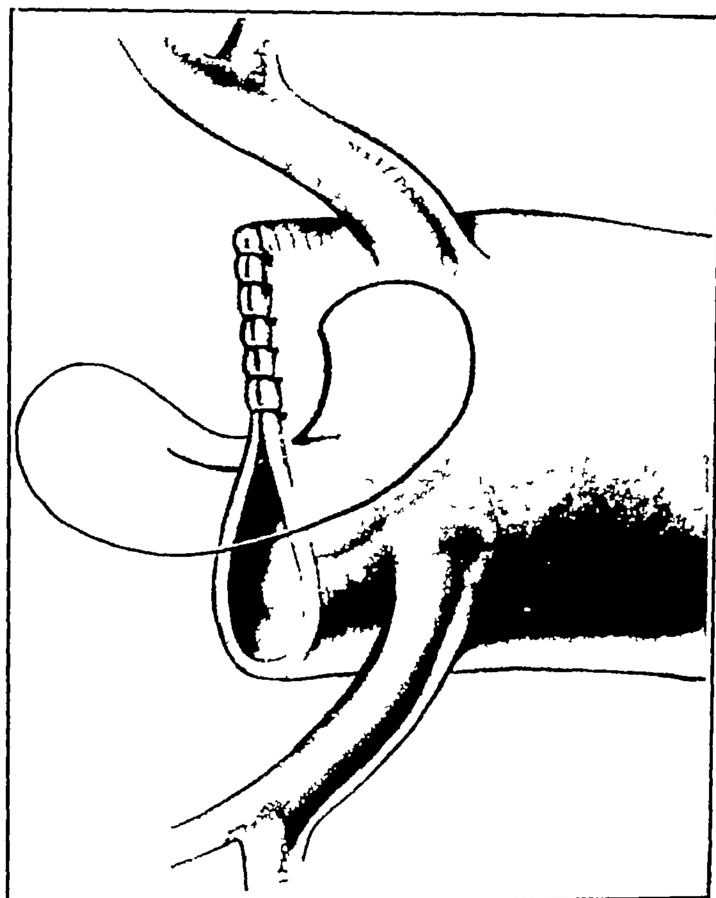


FIG 97 —Suturing of the artery instead of ligating it This saves vessel length, preserves collateral circulation and avoids slipping of the knot

From author's work in Surgery of Trauma Edited by Bowers, W F, Colonel, M C, U S A, Chapter 15, Regional Wound Surgery, Section 2 Vascular Wounds, p 268 J B Lippincott Company, Philadelphia, 1953

from ligature failure or vessel erosion has decreased with this method The advantages of this technic have been confirmed experimentally.[1] It has been used on vessels as large as a patent ductus arteriosus, the aorta, etc [12 29]

Such modifications of standard vessel closure technics should be elected only by experienced surgeons under ideal hospital conditions Locale, combat status or the training of the personnel may make a simple ligation the treatment of choice

*Concomitant Division of Vein When Artery is Resected* – The question of dividing an uninjured vein when an artery has to be ligated has been

argued since Makins report in World War I[17] DeBakey and Simeone[21] apparently have disproved Makins theory of the value of this measure In an injury to an artery however there may be accompanying venous injury or thrombosis For this reason the vein should be opened deliberately and any thrombus removed from the proximal segment to reduce the danger of pulmonary embolism The vein then should be ligated and divided

Vein ligation is always indicated if the vein is damaged but there is not sufficient evidence in the light of our present knowledge that more lives are saved by concomitant vein ligation when the artery must be sacrificed In no instance should further gross dissection be performed to ligate the vein

*Surgical Repair of Arterial Laceration* —The surgical repair of a lacerated artery *at no time* is an emergency or a dressing room procedure nor should it be performed in a front-line dressing post in war time The success of such an operation depends upon surgical sterility trained personnel judgment modern operating room facilities and expert assistance If the patient can be evacuated at once and if the blood supply otherwise would be lost such treatment may be selected The severity of other wounds the length of time ischemia has been present and at times in war or major disasters the number of other casualties may make attempts at such repair impossible

The laceration of the vessel may be complete or incomplete

A INCOMPLETE LACERATION —Where the arterial wall is only partially severed the immediate and visible hemorrhage may not be so severe From the bleeding standpoint such wounds however are more serious than those in which the vessels are completely divided Exsanguination and death may result as shown by Makins[17] and Holman[24] In such cases the wound is held open by the elastic nature of the vessel wall and cannot contract and stop the bleeding Such wounds can be sutured under ideal hospital and personnel circumstances.

*Technic of Repair of an Artery* —The surgical principles outlined for exploration or resection (pages 330 to 331) must be followed After control of the vessels is obtained both proximally and distally all clot is removed and if the repair appears feasible the edges may be freshened The essential principles for successful artery suture are not new and vary little from the dictum of Carrel fifty years ago[17] These are

(a) small-gauge needles and fine silk
(b) avoidance or control of infection
(c) approximation of intima to intima and
(d) gentleness in handling tissues

In addition the vessel should be kept *moist* locally with heparin and saline Hemostasis during suturing can be obtained by rubber bands held with clamps or by rubber-shod arterial or seraphin Snell type clamps A suture is placed at either end of the laceration Gentle traction on these sutures approximates the wound for the repair Two types of suturing are available The use of an everting mattress suture either running or interrupted has been advocated by many surgeons Such a technic sacrifices some of the lumen but is excellent for wounds which are on tension The

simple over and over fine silk suture is an effective and technically easier.[3, 4] If the patient is young, however, and further growth of the vessel is expected, interrupted sutures are indicated at least in part The type of suture and its insertion has to be varied with the situation encountered.

B PERFORATION OF AN ARTERY —Perforation is the second form of wound of a major artery Such injuries are frequently caused by a knife or small missile The bleeding may be severe and often there is an accompany-

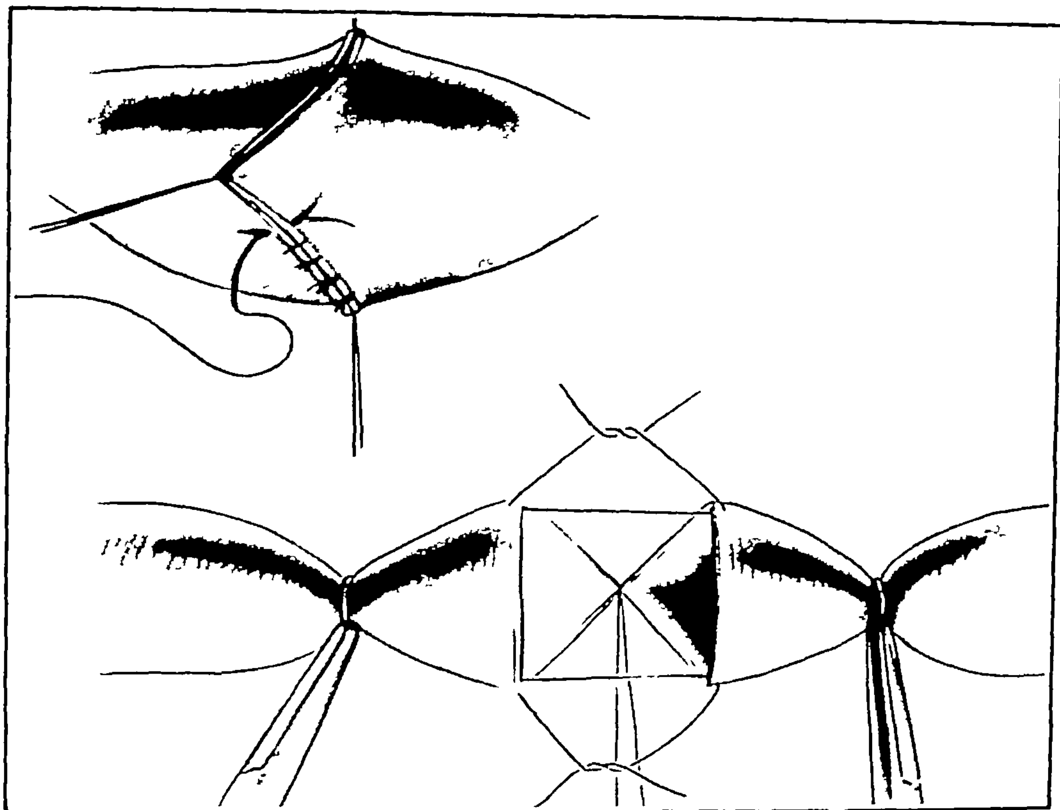


FIG 98 —Triangularization technic of end to end suture Three fine silk sutures are placed equidistant from each other Tension on suture points approximates the edges to be sutured Continuous suture is used where there is no tension Everting mattress sutures are used where there is tension and interrupted sutures in children to permit growth

From author's work published in Surgery of Trauma Edited by Bowers, W F, Colonel, M C, U S A, Chapter 15, Regional Wound Surgery, Section 2 Vascular Wounds, p 268 J B Lippincott Company, Philadelphia, 1953

ing thrombosis There are usually other concomitant tissue injuries In some, the point of ingress may be so small, or the hematoma so large, that the vascular injury is masked entirely While such wounds can be sutured readily, their diagnosis usually is made too late for surgical repair. Rarely, such wounds heal spontaneously, but more often they enlarge as pulsating hematoma or as arteriovenous fistulas if the vein is involved

*Technic of Repair of Perforation* —Operation is indicated once the diagnosis of a perforated artery is made The wound should be surgically cleansed and proximal and distal control of the involved vessel should be obtained before the area is explored The clots are then removed and the

extent of damage determined The vessel lumen is bathed in heparin saline solution and some solution may be left in the involved vessel The wounds are sutured with fine (5-0) arterial silk on eye-sized needles If a pulsating hematoma occurs it is treated as on pages 372 and 380

C COMPLETE LACERATION OF ARTERY —*Complete division of the vessel* is the third type of artery wound There is an immediate severe hemorrhage The vessel usually contracts or is closed by a hematoma and the hemorrhage

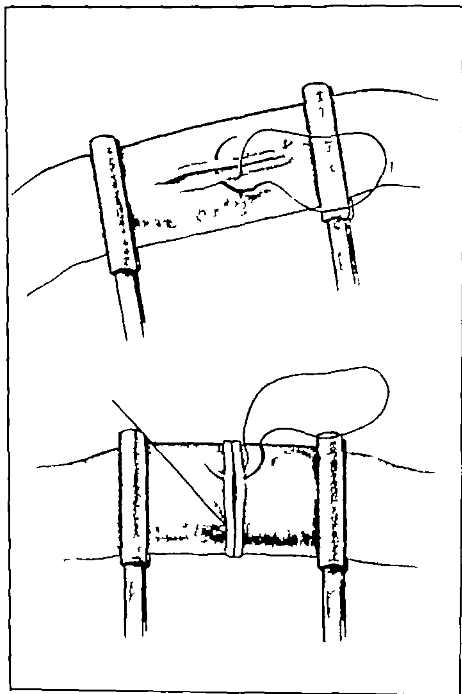


FIG 90 —Arterial suture in which the walls are everted, the intima being brought into contact with intima without foreign body interposition. Such suturing is indicated where there is any tension and is used to bridge gap as in coarctation of the aorta

stops Where a major artery is completely divided, most (but not all) such lesions result in distal gangrene unless there are early facilities for reparative surgery

End to end anastomosis is the ideal treatment in a complete division of an artery The artery is dissected back to obtain the necessary length and nontraumatized vessel ends The vessel may need rerouting or flexion of the joint to decrease the tension on the suture line Where suturing is possible, the triangular technic is performed A simple running over and over suture between the triangulated stay sutures works satisfactorily and is simple technically If the individual is young and vessel growth is expected, interrupted sutures are used An everting mattress suture is used for areas of tension and on large vessels, *i e*, the aorta

*Technic of End to End Arterial Suture* —The modern technic of suturing arteries end to end does not vary greatly from that described in 1896 by Jaboulay[36] and Carrel [17] The ends of the vessel are opposed by three fine stay sutures placed at equidistant points These sutures triangulate the vessel lumen in three straight lines The suturing, whether interrupted or continuous, brings intima to intima on each of the three sides This technic is the same whether end to end anastomosis is performed or grafts are introduced Two types of suture can be inserted The everting mattress suture, whether of continuous or interrupted type, brings intima to intima and is a strong suture if there is any tension The over and over running suture is technically simple and has been employed by Beck,[4] Babcock,[2] Rousselot[62] and the author,[56] as well as others, on many occasions

D Laceration with Loss of Segment of Artery —Severe injuries, or mortar, bomb and shrapnel wounds in war, frequently cause a loss of a section of vessel High-speed travel and industrial injuries combine with diasters to cause such injuries in civilian life The possibility of bridging such a gap depends on the personnel facilities of the hospital, the length of the lost artery, and the time interval In war time the number of other casualties may make such operations impossible The technic of such anastomosis is included for completeness It is stressed again that such operations by unskilled surgeons without adequate equipment, and at the wrong time, may be more disastrous to the patient than if the involved vessel were ligated In such ligations, the collateral circulation may be sufficient to carry on the circulation if the spasm factor is controlled If the artery defect is longer than 3 cm , it can be reunited only by a graft.

*Blood Vessel Grafting* —The replacement of an injured vessel by some type of tube has been considered since the practice of surgery began The use of a foreign tube usually has been followed by thrombosis or rupture and hemorrhage In those instances in which the parts survive, credit had to be given to the collateral circulation The use of *blood vessels* from the patient's own body or from donors has been practiced and is the basis for much of our modern vascular surgery Modern anesthesia technics, antibiotic therapy and anticoagulant drugs have proved the triumvirate which make grafts successful The work of Briau and Jaboulay,[36] Carrel and Guthrie,[18] Watts,[70] Stich,[66] Hufnagel[35] and many others, at least by negative results eliminated many false efforts in this field Crafoord,[21a] Gross,[30] Beck,[5] Sweet,[67a] Touroff,[68a] Holman,[32] Deterling[21b] and others have

contributed greatly in homologous and analogous grafts All types of material have been utilized including gold ivory and vitallium While foreign substances may serve temporarily their use except in an emergency cannot be advised One must expect thrombosis at the point of junction The bridge may work however until the collateral supply is adequate

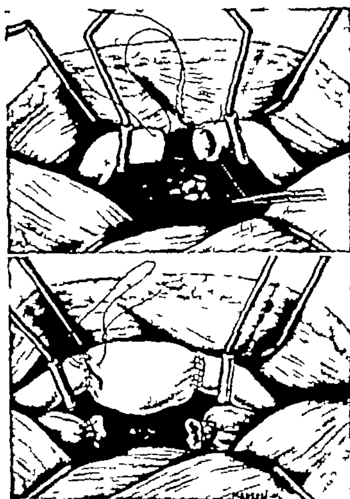


FIG 100 —A. Arterial suturing where there is no tension The suture does not enter the intima and mere approximation of the muscular and adventitial layers is provided. This type of suturing is satisfactory where there is no tension. It is performed after artery exploration.

B Venous transplant Large section of vein has been resected reversed and joined in end to end anastomosis to the artery These large sections of vein will shrink down to the size of the lumen of the arterial flow once the tourniquets are released (Pratt, courtesy of Am J Surg )

*Vein Grafts* —The origin of substituting a vein graft for an arterial loss cannot be dated as it was mentioned by the early Romans who accompanied the Legions The square which was the Roman basic fighting formation was nearly impregnable as long as there were no casualties The loss of a few men when combined with the will of the attacker presented a flank which permitted side attacks defensible only by breaking up the square The laceration of major arteries by a sword or spear thus was an

early battle contingency Earliest efforts to use vein grafts to replace arterial defects were reported by Carrel fifty-one years ago [17] The vein reacts to the arterial pressure by an increase in the fibrous tissue in the middle and inner layers of the outer coats of the grafts [16 31] These findings have been confirmed by many observers [16,18 19 31,70] Our own observations of autopsied specimens have substantiated the fact that veins take over the functions and form of the artery which they replace The author, when associated with Wayne Babcock, observed the effects of the Babcock operation for aneurysm of the ascending aorta [2,2a] In this operation, the distal end of the internal carotid artery is united in an end to end fashion to the proximal end of the jugular vein The vein thickened and appeared similar on section to the artery

## BLOOD VESSEL GRAFTS

**Autografts and Homografts.**—*Arterial autografts* often cannot be used for the obvious reason that they endanger the circulation at the donor site *Venous autografts* have been successfully employed in vessels as large as the aorta Their use in traumatic and military surgery, at this stage, except in rare circumstances, is limited to those patients who can be operated within a few hours' time They can be used for the accidents incident to surgical operations or where growth excision requires sacrifice of the artery This method has obvious possibilities The vein grafts are available readily The saphenous, the femoral, the jugular and the brachial veins, all have been used repeatedly Suturing of the grafts is not difficult technically These vein grafts thicken like the host artery Immediate aneurysmal dilatation occurs occasionally and probably only if there are technical errors in the suturing of the anastomosis The hydrodynamic law, that the wall pressure at any point in a tube of moving liquid is inversely proportional to the rate of flow, applies in such surgery Since the rate of flow is rapid in such an artery, the wall pressure at any point, including the suture line, will be low.

Sufficient numbers of these grafts have been done for there to be some knowledge of their ultimate fate Animal experiments seem to indicate that there will be a dilatation of the vein at the anastomotic line after about a year due to the higher arterial pressure Whether this is valid in humans, with their relatively older vein tissue and less rapid growth as compared to the hog, on which many of the experiments have been done, is not clear The use of the graft is indicated, even if followed later by dilatation, as collateral vessels may form after a time

*Analogous Vein Grafts* —The use of these grafts is feasible in well-equipped hospitals and in the definitive treatment of such lesions as arterial or arteriovenous aneurysms The possibility of their use in early wounds exists but is only to be considered if operation within hours of the injury is possible The type of wound, the extent of the other injuries, the personnel and, to some degree, the number of other wound problems may play a part in the decision to use a graft

*Technic* —The vein selected for the graft should be a nontraumatized one and approximately the same size as the uninjured part of the artery The vena cava has been used as an aorta graft Each branch of the vein should be ligated and transfixed to withstand the arterial pressure The

vein should be immersed in warm saline solution and its proximal and distal ends carefully noted The artery to be grafted should be cleansed and dissected free It is important that the vein be so placed that the distal end joins the proximal end of the artery and vice versa because of the valvular system in the veins The vein and artery are then anastomosed after placing the three triangulation sutures (see pages 335 and 336) A simple running over and over fine arterial silk is usually satisfactory If there is tension or where a large vessel such as the aorta is to be grafted, a mattress everting suture is placed

Interrupted sutures always are used in children The distal clamp should be removed first and then the proximal one Slight bleeding usually will stop with finger pressure Occasionally an extra suture may be necessary but if the suturing technic has been followed carefully this is required rarely Some surgeons reinforce the graft with a plastic material [16]

**Homologous Grafts** —Arterial grafts from other humans have been used many times to bridge defects in arteries due to the loss or disease in a section of the artery The grafts must be from young individuals who are undiseased and must be taken within four to eight hours after the donor s demise Young people who meet traumatic deaths make the best donors There are many problems in getting grafts in civilian surgery including the medicolegal one In military surgery grafts are available due to the autopsy rules

Preservation of Grafts —Gross *et al* [30] originated the preservation of grafts The first method formalin fixation led to fragmentation and calcification of the donor vessel and has been discontinued The grafts can be stored in flasks containing an electrolyte solution (glucose serum) and antibiotics (penicillin and streptomycin) in an ice box at a temperature of 1° to 4° C These grafts have been used successfully after 42 to 45 days In the blood bank in operation by the New York Society for Cardiovascular Surgery (supported by the New York Heart Association) grafts are preserved in a buffered solution to which is added penicillin streptomycin and human serum These grafts were first kept at deep-freeze temperature but in the thawing out some of the cells were destroyed The grafts are best kept at domestic ice box temperature (approximately 2° C ) Hufnagel [illegible] at the National Institute of Health in Washington has preserved grafts in vacuum containers similar to those used for plasma The grafts need not be kept refrigerated have no clotting dangers and keep sterile indefinitely The preparation is complicated at the present time and certain technical difficulties must be overcome This may be the ultimate method of graft preservation

Fate of Homologous Artery Grafts —Animal experiments demonstrated the fate of homologous grafts The fundamental concept that the human body eventually destroys any foreign organic substance introduced into it holds with artery grafts as it does with skin grafts blood transfusions etc The grafts kept in nutrient media survive for as long as forty five days Before this destruction occurs however the host may utilize the grafts as a framework on which to build its own bridge After the graft is in place the cellular elements in the graft are destroyed by the host in three weeks and new intima grows in from the host artery to line the graft At first this growth is fibrocellular—later it assumes an endothelial form and

nonelastic fibrin is formed This growth covers the vasa vasorum as well as the intercostal or other branch vessels A thickened, perivascular coat develops around the outside of the graft The outer part of the graft is revascularized by this heavy coat There is recanalization of the old vasa vasorum vessels in the graft down to the intima This is shown by the fact that if dye is injected, its flow stops just inside the intimal coat The smooth muscle disappears but some connective tissue and elastic tissue remain

TABLE 27 —ARTERIAL ANASTOMOSES IN WAR WOUNDS (WORLD WAR II) RESULTS OBTAINED BY VARIOUS METHODS[65]

| *Method Employed* | *No of Cases* | *Viable Extremity* | *Gangrene of Extremity* | *Gangrene (Per Cent)* |
|---|---|---|---|---|
| Non-suture tube-vein anastomosis (Blakemore) | 70 | 29 | 41 | 58 5 |
| Tube anastomosis | 37 | 17 | 20 | 59 5 |
| Ligation severed artery | 2,655 | 1,355 | 1,300 | 48 9 |
| Suture repair | 141 | 87 | 54 | 38 3 |

TABLE 28 —SITES OF ANASTOMOSIS

| *Number* | *Artery* | *Result* |
|---|---|---|
| 5 | Femoral | Satisfactory anastomoses but only one viable extremity |
| 1 | Axillary (3rd part) | Gangrene of arm |
| 1 | Brachial (proximal to bifurcation) | Arm survived |
| 1 | Popliteal | Death from pulmonary embolism at operation |

Apparently, the inner quarter of the medial coat and the intima obtains nutriment from the blood in the lumen of the vessel The outer three-fourths of the medial layer and the adventitia receive their blood supply from the host This blood supply enters by reopening the lumens of the original vasa vasorum of the graft [40a]

The application and utilization of these grafts appear feasible Grafts cannot be used in emergencies unless trained personnel are available The future of grafts seems assured Their use is indicated in the secondary operations on the circulatory system, the accidental injuries during operations, and on those patients who reach a well-equipped hospital within a few hours after injury The prodecure probably cannot succeed in the presence of gross infection and in war time is *NOT* a front-line procedure The time interval between the injury and the operation is of the greatest importance Recent experiments[19] showed a survival rate in grafts in dogs' legs of 90 per cent if they were applied in six hours, 50 per cent in twelve to eighteen hours, and only 20 per cent if twenty-four hours had elapsed Animal arteries have been employed successfully to bridge gaps, *i e*, pigs'

**Prognosis in Extremity Injuries** —The survival of a limb depends on its blood supply Bailey summarized the incidence of gangrene after ligation of main arteries [3] Other figures are given on page 343, Tables 27 28, 29 and 30.

The experience of the Army Research Team in Korea with 77 major artery injuries shows promise. These patients were operated early, at least within nine hours of their injuries. Only 10 per cent had a major amputation. This figure compares so favorably with previous repair figures as to be revolutionary in arterial repair.[27] (See Tables 24, 25 and 26.)

TABLE 29.—OCCURRENCE OF GANGRENE IN MAJOR ARTERIES[illegible]

| | |
|---|---|
| After Ligation | 57 % |
| After Repair | 52.8% |

TABLE 30.—INCIDENCE OF GANGRENE IN AMERICAN THIRD ARMY DURING WORLD WAR II, AUGUST 1944–MAY 1945

| *Major Blood Vessel Injuries* | *Required Amputation* | |
|---|---|---|
| 837 | 423 | 50% |

In summary, in regard to the replacement treatment of an injury to a necessary artery:

1. Use the injured artery if one can repair or bridge the defect.
2. Use a contiguous vein graft.
3. If available, use an artery bank vessel.
4. Consider autogenous artery (subclavian) or artery from an animal.
5. As a last resort, use some artificial tube or plastic material.
6. Only if the above fails, perform a ligation or suture occlusion.

**Resection of Bone.**—In operating on the subclavian artery or the innominate artery or, at times, on the proximal part of the carotid artery, adequate exposure is again important and resection of the clavicle should be performed. There should be no hesitation to perform this procedure.

The fear that the resection of the clavicle will cause an unstable shoulder girdle has not been borne out by clinical experiences. Resection of the clavicle has been performed since 1859, when Cooper[31] resected the sternal end of the clavicle and part of the sternum in order to approach an aneurysm in the innominate artery.

The ease with which the body adjusts itself to bone loss has been established. Complete resection of the clavicle causes no disability or demonstrable deformity. Such experiences have made the suggestion of Shumacker[44] that the clavicle be chipped and replaced in the periosteum unnecessary. The scapula, large segments of ribs, the fibula, as well as other bones, have been resected and discarded with impunity.

**Suture Material and Suture Technic.**—Despite research in efforts to determine the best suture materials and the most effective suture technic, this work is incomplete and the results at times conflicting. Some studies[19] seem to indicate that catgut is an ideal anastomotic medium for vessels. Catgut sutures are standardized, but the reaction of the body to these sutures varies greatly. A section of catgut may act as a foreign body in one patient weeks after an operation. In another, one may be unable to find any catgut in a wound which dehisces on the seventh postoperative day. Catgut, therefore, cannot be advocated as a routine vascular suture

This applies particularly to traumatic wounds. The use of this suture material in growing children, however, may be considered if the wound is clean and tension is not great. In general, the use of suture material may be summarized as follows:

1. If there is no tension, a simple continuous fine silk suture not entering the intima is advocated for surgical or stab wounds. This may be continuous in adults, but in children interrupted sutures are preferred due to the growth problem.

2. Where considerable growth is to be expected or required and there is no tension, catgut sutures can be used in clean wounds.

3. For simple end to end anastomosis as in severed vessels or artery or vein grafts, *continuous over and over fine silk* sutures are satisfactory. In patients in whom there is difficulty in approximation, a continuous mattress everting suture works as well. It is extremely valuable in adults.

4. Where growth is expected and tension exists *interrupted everting* mattress *silk* sutures are best. These should be inserted at approximately 1 millimeter apart. If technically difficult only one-half of the sutures need be interrupted.

5. The use of a foreign body tube, whether endothelially lined or not, is a temporary expedient. Thrombosis may occur and usually rupture or necrosis will take place at the site of anastomosis. In this respect, Nature follows its usual path. A pulsating blood vessel as it enlarges will erode resistant structures instead of pressing soft tissues. Thus, we see the erosion of the vertebrae or sternum by an aortic aneurysm. Success after such foreign tube anastomosis will depend upon the development of collateral circulation and unless thrombosis occurs at the site of anastomosis, erosion is likely. As a temporary bridge such a tube works well.

If there is slight leakage at the suture line, this can be eliminated by mild pressure with a moist sponge or the gloved finger. The pressure within the vessel is inversely proportional to the rate of flow and therefore very little tendency to leakage is present provided there is an adequate channel for the blood to follow.

**Postoperative Care.**—After surgical treatment, the part is immobilized and placed at heart level, or slightly below, and precautions taken to protect it from local injury by pressure or thermal changes.

**Other Therapy.**—(1) *Anticoagulant therapy* is regulated by daily laboratory reports. The danger of bleeding from the suture site as a result of the use of anticoagulants exists but usually is controllable. The dosage, therefore, must be carefully watched and directed depending on laboratory reports. The degree of success is substantially greater when *anticoagulant* therapy is used to the therapeutic level. This treatment should *not* be neglected or delayed. Venous thrombosis also will be minimized in this way. For further discussion, see chapter on Antithrombotic Therapy, pages 651 to 666.

(2) *Sympathetic nerve blocks* with novocain should be repeated as often as necessary immediately after the operation in order to relax the spasm in the operated and collateral vessels. In many instances the life of the part can be retained by this procedure. In others, sympathectomy should be performed, but in serious cases this may be delayed or replaced by sympa-

thetic nerve blocks If sympathetic blocks cannot be performed due to technical difficulties medical sympathetic blocks are advocated Intravenous procaine ether intramuscular tetraethylammonium chloride or Priscoline may be used

(3) *Antibiotic therapy* including sulfa drugs penicillin streptomycin, etc., as well as gas gangrene prophylactic antiserum, should be given to control infection

## LACERATED ARTERIES IN INFECTED WOUNDS

Many lacerated vessels in infected wounds can be re-established and the infection controlled with antibiotic therapy In the presence of infection the decision as to blood vessel anastomoses must be individualized Many gunshot wounds from the last war and the Korean incident did not even suppurate and the early practice of opening such wounds soon was discontinued Thus antibiotic therapy and the anticoagulant therapy has changed our entire conception of vascular surgery after traumatic wounds

In grossly contaminated cases the wound should be managed surgically the same as any other traumatic wound All devitalized tissue is removed all pockets are opened antibiotics should be used and the wound left open The administration of antibiotics, as well as gas gangrene antiserum is fundamental and should be given in every case Immobilization of the part will help in the healing and in localizing any infection

Experimental surgery has shown how well the blood vessels tolerate repair even in infected wounds End to end anastomosis arterial repairs and even vein grafts healed in grossly infected wounds without an arterial blow out or arterial hemorrhage [illegible] In this work if heparin was added to the blood stream these vessels continued their patency and carried their blood load Such findings have contributed vastly to our knowledge of reconstructive vascular surgery in the presence of infection and have served *to establish the value of anticoagulants in maintaining the circulation*

## FOREIGN BODY TUBES FOR REPLACING LOST PARTS OF ARTERIES

Modern technics of using some artificial material to replace the lost part of blood vessels or to facilitate repair followed the early attempts of surgeons to transfuse blood The same aids developed for blood transfusions were later applied to anastomose blood vessels

**Early Cannulae** —Crile [22] as early as 1898 used an artificial bridging method His cannulae were of metal with flanges His technic consisted of pulling the vessel through and doubling it back on the cannula thus establishing an endothelium-lined tube In blood transfusions Crile pulled the artery over the reflected vessel and then tied it over a flange of his cannula avoiding suturing and its attendant thrombosis

Four years later in 1902 Landon[41] also devised a cannula based on Crile s endothelium lined technic of anastomosis.

Various other types of cannulae were later developed by Brewer [13] Bernheim [6] Bryan and Ruff [14] and Elsberg [27]

The first to use vein cannulae were Dorrence and Ginsberg.[25] Curtis and David[23] used a Y-shaped cannula. Tuffier[68] used silver tubes, and Kimpton and Brown[41] introduced glass tubes coated with paraffin.

**Vitallium Cannulae.**—Technical difficulties with suture anastomosis in such work as portacaval and other shunts caused a revision of this non-suture technic in 1945. Blakemore and Lord[8,9] used vitallium tubes and endothelially lined them by pulling the end of the vein through the tube and doubling it back on the tube over a flange. The other vessel is then pulled over the first and the anastomosis is held together by ties applied over the tube. The flanges prevent the ties from slipping off.

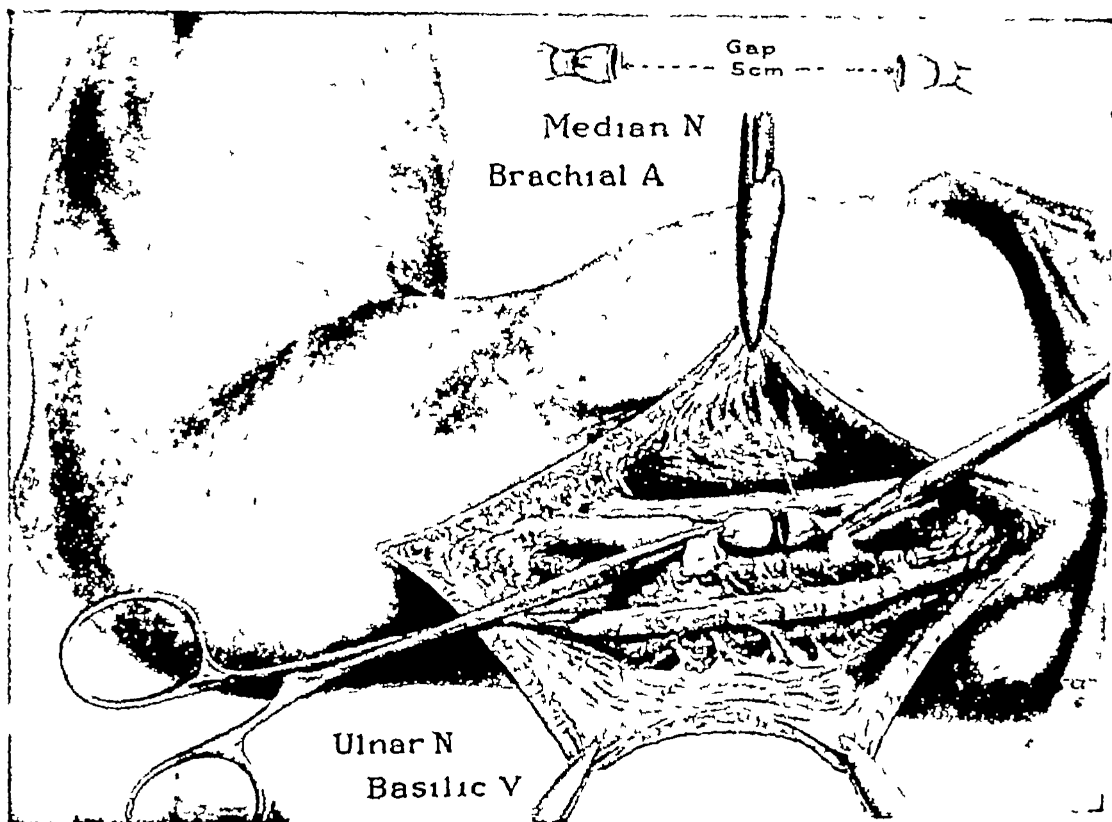



FIG. 101.—End to end arteriorrhaphy of the brachial artery. The aneurysm has been excised. Apposition of the divided ends is obtained by flexing the elbow and by gentle traction upon the vessel. Hemostasis by tapes wrapped around the artery and held by hemostatic forceps. (Courtesy of Babcock's *Principles and Practice of Surgery*, Lea & Febiger.)

This technic will bridge a gap in a ruptured blood vessel, at least temporarily, when suturing is not feasible, technically impossible, or contraindicated for some reason such as tissue loss. It has the disadvantage, however, that rupture may occur at the point where the tube ends. Thrombosis is another complication. Callow *et al.*[15] reported that every vessel so joined, in their experimental animals, thrombosed.

**Plastic Tubes.**—Plastic tubes to which each end of the divided artery is tied have been used to bridge artery lacerations. When collateral circulation has developed, this tube is removed and the vessel is ligated. This second step is necessary to avoid the slough and hemorrhage that develops

at the point of anastomosis of the fibrin tube and the blood vessel if the tube is not removed In experiments on animals thrombosis occurred very early in every limb so anastomosed [15]

**Tubes versus Suturing** —The use of a tube such as the one described by Murray and Janes [11] or that of Blakemore and Lord[1] (see page 346) has been advocated on the grounds that their use is technically easier than suturing

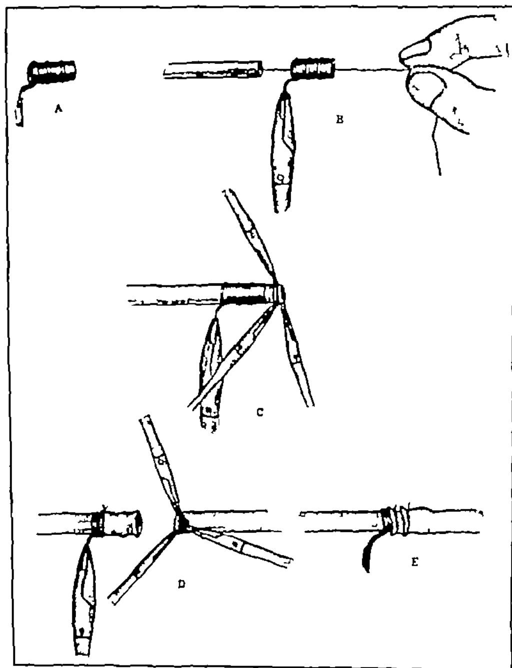



FIG 102 —Crile cannula. Technic devised by Crile Threading a vein through a tube and cuffing it back to form endothelial lining. A. Crile cannula. B Thread in the vein to draw it through cannula. C The vein is caught by three mosquito hemostats and cuffed back over the cannula, and at D is tied over the ridge next to the handle The artery is slipped over the cuffed vein and fastened with a suture on the ridge farthest from the handle E The operation completed. (Horsley Surgery of the Blood Vessels courtesy of C V Mosby Company 1915)

The contention that suturing causes more foreign body reaction and, therefore thrombosis, is not valid. Only in emergencies or where suturing is impossible should the use of a tube be considered in lieu of suturing. That suturing is the ideal type of anastomosis is testified to by others[11,15,24,30,39,45,49,51,63,65]

It should be emphasized that the use of any rigid tube or foreign material, regardless of whether it is endothelium-lined or not, definitely increases the danger of slough and necrosis or thrombosis at the tied area, with the increased wall pressure at that point.

Animal observations confirmed this point. Investigators[51] noted that when metal clips were used to anastomose severed arteries of dogs, an excellent arterial union occurred in every case, but the animals all died of hemorrhage from two to six weeks after the anastomosis. The site of the hemorrhage was immediately adjacent to the site of the clip, where necrosis of the arterial wall had occurred. This necrosis did not occur when anastomosis was performed with fine silk.

Ligation also acts much in the same way. Biggers,[7] Matas,[48] Appleby and our own Clinic have found that when a large vessel such as the aorta is ligated in continuity, such patients frequently die of hemorrhage, which occurs with a blow-out just proximal to the point of ligation.

If used, the tube should be removed subsequently with suturing or by ligation of the vessel. *If rupture or thrombosis does not follow the use of a foreign body tube, one must assume that collateral circulation deserves the credit. If a foreign material tube is used for anastomosis, it should be considered only as a temporary measure.*[61]

A review of the literature on attempts to ligate the aorta in human patients reveals that 8 of 36 survived one year. The rest of these subsequently died, with the exception of 1 patient recorded by Reid.[59] *In this patient, Reid placed a fascial graft or plug of fascia or other substance proximal to the point of ligation of the aorta, thus making a long thick area of thrombosis proximal to the point of actual ligation of the vessel. This area thus accepted and absorbed the arterial shock. One of my patients, similarly treated, is alive after four years.*

*It can be stated, on the basis of these reports, that when a restricting force is applied to the arterial side of the circulation, if thrombosis does not occur a good distance proximal to this constriction point, rupture and hemorrhage will develop inevitably.*

**Other Tubes and Materials.**—In addition to the vitallium cannulae and the plastic tubes of Murray and Janes, endothelium-lined fibrin tubes have been used by Swenson and Gross.[67]

The possibility of using some absorbable material, such as glucose, which will dissolve in the blood stream after it has served its purpose, has been considered. It acts as a darning ball type of structure to keep the lumen the right size while suturing is being performed. This technic has been used experimentally, but at present does not have clinical application.

With the great progress in development of various plastic materials, the possibility exists that one or several of these may be used to replace a lost vessel. Blakemore *et al*[69] experimentally has employed a plastic cloth called Vinyon "N".* It has long been our feeling that any *foreign*

* Union Carbide and Carbon Corp., New York, N. Y.

substance which replaces a segment of an artery must have expansile properties to absorb the thrust caused by cardiac systole A rigid substance no matter of what it is composed will erode at the junction point between the blood vessel and the foreign substance due to the force of the blood flow The ideal substance must be capable of temporarily enlarging as do the human arteries without a permanent dilation Several such substances are currently under investigation

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# Chapter 19

# ARTERIAL THROMBOSIS

*Acute Arterial Thrombosis Not Due to Trauma.*
*Arterial Thrombosis Due to Trauma*

ARTERIAL thrombosis may be due to trauma or disease. The signs and symptoms and to some extent the therapy for the thrombosis due to trauma have been detailed in Chapter 18 (see pages 321 to 351). Acute arterial thrombosis occurs in the absence of trauma in diseased vessels and, most often, in vessels in which the calcium deposit is in the form of plaques.

## ACUTE ARTERIAL THROMBOSIS NOT DUE TO TRAUMA

**Etiology.**—In diseased vessels in the absence of trauma, acute arterial thrombosis also may occur from arterial stasis, especially in the presence of mild trauma. Arteries which are diseased are subject to an acute closure if stasis or some change in the cellular components of the blood occurs. Such vessels, in addition, may occlude suddenly, due to a clot formation at any site of endothelial disruption. A trauma so mild as to be irrelevant in the normal vessel may be the instigator of a complete occlusion in a diseased vessel. Such an injury may be one precipitated by bed rest, the two layers of endothelium being bruised slightly by pressure alone.

Conditions which tend to increase the relative cellular content of the circulating blood, such as erythrocytemia, marked dehydration, anemia, hemorrhage, blood transfusions, malaria, malignancy, obesity, venous stasis, and shock, predispose to arterial thrombus formation.

In most cases, the thrombosis may be of insidious origin. The vessel distal to the clot may carry blood satisfactorily through the reduced lumen until such time as the thrombus completely occludes it. That the thrombosis may occur primarily without embolism is a poorly understood fact. In most instances of occlusion at a major bifurcation, the resulting symptoms are interpreted as of embolic origin. The differential points between embolism and thrombosis in the aorta were first described by Graham in 1814.[5] The first reported diagnosis was that by Barth in 1848.[4] In his patient, there was a clot from the aortic bifurcation to the superior mesenteric artery. A small channel permitted blood to pass, but the symptoms of numbness and intermittent claudication progressively appeared. Welsh's report[18] that 11 out of 59 occlusions at the bifurcation of the aorta were of thrombotic rather than embolic origin gives us a figure of 23 per cent incidence. From 20 to 40 per cent of all acute arterial

closures probably are due to arterial clotting. The most serious arterial thrombosis with which we have to deal is in the end vessel of the mesentery, the major vessels of the heart, those in the brain, and at the bifurcation of the aorta. This latter group were adequately described by Leriche,[12] and though the description followed essentially that of Welsh, all of Leriche's cases were proven at operation and the syndrome of the thrombosis at the bifurcation of the aorta in general bears his name. An arterial bifurcation is a natural site for thrombosis since at such points the stream is diverted and the speed of the flow is slowed by the narrowing of the distal vessels. Peripheral resistance is increased and there is some trauma to the blood vessel wall. This latter trauma may initiate the disturbance in the intima and permit deposits of cholesterol and calcium. With the continuation of the process, the intima may become roughened and ulcerated. These changes are observed more often if there is a disease of the walls such as arteritis, various types of aneurysms, or a congenital narrowing such as in coarctation.

A brief resumé of nontraumatic arterial thrombosis follows.

In some patients with symptoms of thrombosis at the bifurcation of the abdominal aorta, the clot may originate in the femoral or iliac arteries and propagate proximally. If this extends high enough, the renal arteries may be involved and the terminal stage then may be uremia.

Arteriosclerosis, atherosclerosis and thromboangiitis obliterans produce the underlying pathologic lesion with thickening or partial obliterations of the lumen. The blood stream thus slows and any alteration which results in stasis, a change in the clotting factor or disturbance of the intima may cause the final occlusion. The usual causes of arterial thrombosis are listed below. The pathogenesis of nontraumatic arterial thrombosis is of two types. An acute occlusion may follow the undermining of a large plaque. In such cases, the collateral blood supply usually is inadequate and the same acute symptoms develop as in traumatic thrombosis. If the collaterals are not adequate and nothing surgical can be performed to remove the block, ischemia and gangrene will be the result. In the other type, the lumen has been obliterated slowly by the arteriosclerosis or medial atherosclerosis with a partial occlusion occurring over a period of years. Such a slow occlusion stimulates the collateral circulation. The final acute thrombosis may cause a lack of blood supply temporarily followed by the resumption of this blood supply by collateral circulation. Gangrene is rare in such occlusion.

*a. Infection as a Cause of Acute Arterial Thrombosis.*—There are three methods by which infections can cause acute arterial occlusion. The diseases pneumonia, influenza, typhoid fever, ulcerative colitis as well as other septic infections, have been known to cause occlusions. An infectious disease may also directly invade the arterial system. In such cases there may be so much change in the blood supply as to cause the artery to close. In a third way, the inflammation due to an acute infection may produce so much edema and venous stasis as to occlude the arterial supply secondarily.

*b. The Blood Dyscrasias as a Cause for Acute Arterial Thrombosis.*—The blood dyscrasias such as polycythemia vera, anemia or chlorosis may

cause an arterial thrombosis In addition, venous thrombosis alone has caused an occlusion The cause for the arterial thrombosis is an increased coagulability of the blood and an increased tendency to clot due to a change in the intravascular clotting factor

c *Congestive Heart Failure or Cardiac Insufficiency as a Cause for Acute Arterial Thrombosis* —The blood stream slows in congestive heart failure This decrease in the circulation is particularly noticeable in the terminal stages There may be an increased coagulability of the blood In addition, patients with cardiac insufficiency have a tendency to arterial embolism.

d. *Fungus Infection as a Cause for Arterial Occlusion* —Very little is known about the way fungus infection passes through the vascular system The fungus organism is believed to pass through the vascular system either actively or in one of the "tid" forms The occlusive reaction may be on the allergic or spastic basis Usually only a small vessel is involved

e *A Slowed Blood Stream as a Cause for Acute Arterial Occlusion* —A reduced circulation may contribute to arterial thrombosis Any such condition as shock, hemorrhage, hypotension, visceral damage, narcotics, inactivity produced by various casts and orthopedic appliances and bed rest itself may result in an acute arterial thrombosis This occurs most often if the patient has some underlying arterial disease

f. *X-ray, Radium or Radioactive Isotopes as a Cause for Acute Arterial Thrombosis* —The slowed blood stream after intensive ray therapy may initiate an acute arterial thrombosis

*Trauma* itself may precipitate the thrombosis in diseased arteries This point is important, with the more liberal interpretations of the compensation laws to include not only injury but occupational diseases For further details, see chapter on Cardiovascular Diseases and Trauma, page 804

*Significance of Mild Trauma on the Patient with Diseased Arteries* —The part that a mild trauma may play in aggravating or accelerating the occlusion process in the diseased artery is of medicolegal importance Each case must be decided specifically on its merits, but certain underlying principles might well be stressed A vessel with atheromatous changes and calcification in the form of beads or plaques may occlude suddenly and acutely sometime in the patient's lifetime These occlusions have occurred while the patient was asleep, walking, or working

In order for the alleged trauma to be called the exciting or aggravating cause of the acute closure, certain premises must be fulfilled

(1) The occurrence of trauma of sufficient severity to have caused the occlusion must be established

(2) The occlusion must have occurred at or contiguous to the site of trauma

(3) The symptoms of occlusion must have dated from the time of the trauma and the progress of the disease must have changed markedly from that moment

(4) Previous episodes of similar occlusion without trauma must be excluded

(5) A severe strain may produce an acute occlusion This strain must be of an unusual type A violent pulling or jerking in an awkward or ungainly position, in exceptional instances, may cause a thrombosis A

strain which increases the intraarterial pressure may loosen a plaque and obstruct the vessel This is of rare occurrence

**Symptoms** —Where there has been a previous arterial disease, *claudication* may be already present. With this blood deprivation, there will be *trophic changes* in the digits or nails All the symptoms detailed under arteriosclerosis may be present

Where the collateral circulation has been adequate, there may be no symptoms prior to the sudden occlusion The symptoms then are of acute arterial occlusion The symptoms depend on the site and degree of the thrombosis

With an acute arterial thrombosis the part becomes *pale white cold* and *paralyzed* *Pain* appears at the site of the clot and may be due both to presence of the clot and spasm

*Venous congestion* follows with a skin mottling and duskiness If the collateral circulation is insufficient or remains in spasm the *mottling* turns to a purple color and a line of demarcation distal to the occlusion point soon appears In such event the pain persists as does the coldness and paralysis In others where the collateral supply is adequate the mottling may be followed by a rubor and in time a partial or complete return of circulation

Venous thrombosis usually will occur if the occlusion persists

The diagnosis of arterial thrombosis depends upon the site of the occlusion No attempt is made to detail the symptoms of such obvious occlusions as the cerebral or the coronary arteries. Symptoms of mesenteric thrombosis are given on pages 405 to 406 The diagnosis of closure of the major vessels will be divided into those at the bifurcation of the aorta and those in the arteries below the iliac or subclavian arteries

1 *Thrombosis of the Bifurcation of the Aorta*—The symptoms depend upon the acuteness of the closure. The longer the symptoms of partial occlusion have occurred the less severe will be the signs of the final picture Thus, in Elkin s and Cooper s[3] 10 patients, the 2 patients with the shortest histories developed the most severe symptoms and serious trophic changes the most rapidly The earliest symptoms are intermittent claudication fatigue, numbness, coldness of the part and pain beginning in the hips back or pelvis and extending down through the calves Trophic and color changes depend upon the degree and rapidity of development. The classical symptoms of arterial occlusion such as rubor on dependency and pallor on elevation loss of hair atrophy, and eventual ulceration and gangrene may be expected A recent patient referred to the author is an example of the sudden occlusion

*Case Report* —The patient, age forty two had been working as a fruit stand attendant up to two days prior to his admission to the hospital as an emergency Gangrene was present in the left leg and extended despite extensive lumbar sympathectomy Amputation was performed in the lower third of the thigh but necrosis of the stump required re-amputation the next week near the hip Due to occlusion signs in the other leg extensive lumbar sympathectomy on the right side also was performed The second amputation stump did not heal and necrosis began on the right foot a week later Amputation on the right leg was performed but resulted in nonhealing The gangrene extended proximally with necrosis of all of the skin

*on the buttocks, lower back and pelvis. This exposed sloughing muscles and fascia. All types of supportive therapy were utilized. The patient survived for eight weeks, with no evidence of viability of either leg, hip or back before death intervened. See Figure 57, page 212.*

This is an example of a patient with an acute thrombosis of the bifurcation of the aorta which propagated into all collateral vessels. Bilateral lumbar sympathectomy made no change in the extension of the disease. In this instance, an apparently well individual was struck down by a fulminating thrombosis which all known therapy including antithrombotics could not retard. From the time of admission, the patient's general status never appeared satisfactory enough to consider resection of the aorta.

2. *Thrombosis of Arteries Distal to the Iliac or Subclavian Artery.—In the lower extremities*, the diagnosis is based on the symptoms of failing arterial circulation. These again vary with the rapidity of the onset and the propagation of the clot. The diagnosis is made on the symptoms of arterial failure.

*In the upper extremities* there may be an initial state of spasm of the collateral vessels. Since the blood supply is more adequate in this area, relief of the spasm may be all that is necessary. The differential points to be elucidated must be considered to rule out embolism or venous thrombitis, with reflex arteriospasm. The following points must be considered under differential diagnosis.

**Diagnosis.**—Arterial embolism and thrombophlebitis must be differentiated from arterial thrombosis.

(1) *Embolism.*—Ninety per cent of the cases of embolism have left-sided heart disease, with auricular fibrillation. The occlusion in embolism is more sudden and complete, and is usually at the bifurcation of the artery. In arterial thrombosis, a roentgenogram may reveal sclerotic changes in the thrombotic vessel. In embolism, the artery is previously not diseased, nor is there evidence of lesions in other arteries.

(2) *Thrombitis (Thrombophlebitis).*—If spasm is an important factor in thrombitis it may have to be differentiated. In thrombitis, the major arteries are palpable, unless temporarily in spasm. The part is warm, congested, and swollen. Sometimes, sympathetic nerve blocks may be necessary to differentiate between the conditions.

The varying points in the diagnosis of embolism, arterial thrombosis and thrombophlebitis are summarized in Table 30.

**Treatment.**—The treatment of arterial thrombosis of the mesenteric vessels is considered under the chapter Arterial and Venous Occlusion of the Mesenteric Vessel (see pages 464 to 469).

1. CEREBRAL ARTERIAL THROMBOSIS.—Treatment of occlusion of the cerebral vessels includes therapy for the underlying disease and the symptoms as they develop. Since many of the early findings after such a brain insult are due to spasm, its relief is advocated and may be life-saving. Later the edema and anoxemia add to the spastic symptoms.[illegible] Further evidence that these factors are important is the transient and fleeting changes and sudden recovery or extension of the lesions seen so often clinically. Stellate sympathetic nerve blocks have become routine therapy in some patients whether the lesion be thrombotic or embolic. The techni-

of this is simple the armamentarium is always available and any well trained surgeon internist or anesthetist can master it with study on the cadaver The possibility that such therapy might increase the signs or be fatal if the lesion is a cerebral hemorrhage is not tenable and should not restrict this therapy Cerebral hemorrhages of any magnitude are associated with hypertension and their prognosis is poor with or without any therapy Differential points between embolism thrombosis and hemorrhage often are impossible One may follow an old dictum which stated that if the patient recovered it was a thrombosis or embolism and if he died it was hemorrhage One should not deprive the thrombotic or embolic patient of this therapy merely because the primary lesion may be a hemorrhage To these blocks as in all with arterial thrombosis should be added

TABLE 30 —DIFFERENTIAL DIAGNOSTIC POINTS IN EMBOLISM ARTERIAL THROMBOSIS AND THROMBITIS

| | *Embolism* | *Arterial Thrombosis* | *Thrombitis* |
|---|---|---|---|
| Etiology | Left-sided heart disease auricular fibrillation 90% | Arterial disease and/or trauma | Postoperative post partum chemical or trauma fungus |
| Symptoms | Coldness pain, no pulse auricular fibrillation | Coldness pain no pulse | Heat duskiness swelling fever |
| Diagnosis | Made on etiology symptom and signs | Roentgen ray claudication etc | On symptoms and normal pulsation of artery |
| Oscillometric Readings | Zero | Zero | Normal unless in spasm |
| Roentgen Ray | Negative | Usually positive for arteriosclerosis | Negative |
| Age | Any age but usually 20 to 40 years | Usually over 45 years | Any age |
| Skin Temperature | Cold | Cold | Hot |
| Color | White or mottled | White or mottled | Dusky cyanotic to red |

anticoagulant therapy General supportive measures are necessary In some the intravenous use of procaine has been tried but the results are not comparable to the effect of blocks The blocks should be continued until a definite outcome has occurred The anticoagulant therapy should be maintained for weeks or months and under controlled conditions perhaps indefinitely See chapter on Antithrombotic Substances Urinary incontinence and other symptoms should be treated as they arise

2 CORONARY THROMBOSIS —The therapy of arterial thrombosis in the renal or coronary or other inaccessible vessels in the light of our present knowledge is conservative Thrombectomy in the acute coronary occlusion has appealed to the author, but the patient and the surgical preparation have never appeared coincidentally

3 TREATMENT OF THROMBOSIS OF THE BIFURCATION OF THE AORTA —(a) *Thrombectomy or Grafting* —The ideal therapy would be to remove the thrombosis or resect the thrombosed vessel The latter measure re-

quires heroic surgery, and since these patients have advanced disease with acute closure of the aorta it rarely is feasible The vessels do not lend themselves well to suturing The number of patients with such lesions in any surgeon's experience is not great The feasibility of anastomosis without suturing has appealed to many Such anastomosis must be made with some nonrigid material to avoid the inevitable rupture which follows the insertion of a solid foreign body in a pulsating vessel It is conceivable that this problem may be overcome Blood vessel grafts of homologus or anologous types are available, and if one can overcome the technical problems of anastomosis of diseased vessels this may lead to the solving of this problem

(b) *Lumbar Sympathectomy* —This operation has been performed for thrombosis of the aortic bifurcation by many authors [3,12,13,19] The author has utilized the method in 18 patients with survival in 10 Because of the advanced nature of the disease one cannot expect better than 65 per cent recovery The possibility of extending the thrombus or having it become embolic during the operation must be kept in mind and has been emphasized on page 510 The light hand on the retractor, a nontraumatic exposure and an extensive excision of the ganglia chain will be followed by more recoveries One should not try to demonstrate the pathology or the anatomy of this sympathetic system to an assistant or interested bystanders on such patients

(c) *Operative Resection of the Bifurcation of the Aorta* —Many years ago Leriche emphasized that a thrombosed vessel was no longer a part of the arterial system With its occlusion it becomes a carrier of vasoconstriction impulses It also originates reflex arteriospasm To these points, however, must be added the most important one The continued presence of a thrombosed vessel may be the cause of an extending thrombosis proximally, or distally, and into vital organs Embolism from such a site also may occur at any time

Excision of this bifurcation is a formidable procedure The aorta cannot be clamped as the arteriosclerotic part will fracture In many the aorta is like a chicken's trachea with varying sized bone-like projections caused by the plaques Such an organ cannot be sutured In one instance, the author introduced fascia as a pad to take the pounding of the proximal pulsation off a suture line It is suggested that the lumbar sympathectomy in such cases be performed transperitoneally and the condition of the aorta ascertained If operation and resection are feasible then it can be performed Since many of these patients do well on lumbar sympathectomy alone, this should be the first step If the condition progresses despite this measure the decision to try to resect the bifurcation will depend upon whether it appeared feasible when the organ was inspected during the sympathectomy

4 TREATMENT OF THROMBOSIS FOR DISTAL VESSELS —If these lesions are seen early, lumbar sympathectomy is the treatment of choice followed by anticoagulant therapy Failure to remove the source of occlusion will be followed by propagation of the clot in most cases. Since most of these patients are seen late after an insidious onset, therapy should be surgical sympathectomy followed by the anticoagulant drugs To this

should be added all of the measures described under the surgical management of acute arterial occlusion (See pages 453 to 463.)

(a) *Arterectomy or Intimectomy*.—As was described under thrombosis of the bifurcation of the aorta, a thrombosed artery is no longer a part of the positive circulatory system. Its resection therefore is feasible and removes the origin of propagating clot. In addition it negates the reflex arteriospasm arising from the thrombosed vessel. An arterial sympathectomy also results from such a procedure. This operation has been described in detail under arteriosclerosis. (See pages 205 to 210.)

(b) *Endarterectomy*.—Thrombosis may cause the death of the lining layers of a vessel while the adventitia may remain viable. The excision of such 'dead' layers has been advocated and is feasible in selected instances. The number of patients on whom this operative technic can be tried will increase with the more general use of arteriography.

(c) *Vessel Grafts*.—The use of analogous vein or homologous artery grafts to bridge areas of thrombosed arteries has been discussed on pages 203 to 205. This method obviously has definite possibilities and in many instances, especially in the young individual, it has a place in the therapy.

**Antithrombotic Therapy in Nontraumatic Thrombosis**.—To these measures anticoagulant therapy should be added. This should be to a therapeutic level and continued for an indefinite time. Such therapy will become more practical with the development of better tests for prothrombin and better drugs in the future.

If the outlined therapy fails, the limb is permitted to demarcate completely. When demarcation is complete, amputation may be necessary. This amputation should be delayed to permit complete separation and the development of collateral circulation, unless there is sepsis. In the absence of infection, amputation delay is sound therapy. Where uncontrolled infection complicates the picture, early amputation may be required.

Penicillin, streptomycin and other antibiotics as well as gas gangrene antiserum should be administered in efforts to control or prevent infection.

In addition, all the methods of care of the limb detailed under occlusive arterial diseases (see page 164) should be followed. These include (1) avoidance of spasm, *i.e.*, no smoking, ergot, etc.; (2) good hygiene and care to avoid skin breaks or trauma or infection of the part; (3) efforts to stimulate collateral circulation by soaks, antispasmodic drugs and interruption of the sympathetic system.

## ARTERIAL THROMBOSIS DUE TO TRAUMA

**Etiology**.—Trauma is a usual cause for arterial thrombosis in a normal artery. Such trauma may be the result of a laceration or contusion. In such instances there is a direct injury. A hemorrhage or subcutaneous hematoma may develop.

There may also be an indirect trauma to the artery which will cause a thrombosis to develop. There is usually direct injury to the other tissues rather than the artery and the thrombosis of the artery is secondary to pressure either indirectly at the time of the injury or from the reaction to the injury. The result of either of these causes will be evidence of in-

adequacy of the arterial supply distal to the point of injury. The part will be white, pulseless and it may be paralyzed, depending on the site of the thrombosis. There will be ecchymosis at the point of the injury. Venous stasis develops distal to this area and this may increase. Cyanosis will develop if the collateral circulation is not capable of carrying on for the thrombosed vessel. Ischemia will follow and there will be a line of demarcation and gangrene if it is unrelieved.

*Secondary Trauma or Compression as a Cause of Acute Arterial Occlusion.*—A compression without direct injury to the vessel may cause sufficient injury to the lining of the vessel as to cause it to occlude.

*Obstetrical Delivery as a Cause for Arterial Thrombosis.*—During obstetrical delivery, the pressure of the baby's head or other parts of the body may compress a major vessel and cause arterial occlusion. This may occur also on the basis of reflex arterial spasm. The possibility of an embolic origin of the occlusion must be considered. This may occur from the heart, from an area in the artery, or paradoxically from venous pathology.

*Acute Arterial Thrombosis Associated with X-ray, Radium or Radioactive Isotopes.*—Any of the radioactive and radiotherapeutic measures may cause an acute arterial thrombosis. These agents may act again by slowing the blood stream or traumatically causing sufficient pressure to allow thrombosis to occur. Scarring and retraction may cause the syndrome.

*Frostbite as a Cause for Arterial Occlusion.*—If one is exposed to a low temperature for a short time or to a moderately low temperature for a long time, acute arterial occlusion may occur. The severity of the arterial occlusion has caused frostbite to be divided into stages similar to that of burns. The frostbite which occurs with anoxemia of high flying is an important cause for acute arterial thrombosis.

Arterial thrombosis may be due to trauma alone and may occur in the absence of arterial disease. This has been discussed in part in Chapter 19, pages 359 to 362. When arterial thrombosis is due to trauma, it occurs at the site of or in close proximity to the point of injury. Thrombosis may be due to direct injury of the artery, or it may be due indirectly to pressure. The endothelial walls may be bruised at the time of injury with intimal damage. The thrombus forms. It then may propagate.

**Symptoms.**—The symptoms vary with the site of the thrombosis. There is usually external evidence of the injury by continuous internal or external *hemorrhage* and *swelling*. The extremity distal to the point of injury first *blanches* and then later *mottles* and becomes bluer as the blood collects in the veins in the absence of arterial blood to move it through the capillary bed. There is *paralysis* of the extremity, unless some of the muscles above the arterial injury point have a distal function. The *pain* may be extreme and will be greatest at the site of the injury. Later, there may be *anesthesia* and *paresthesia*.

**Diagnosis.**—The diagnosis and differential diagnosis have been covered on page 357.

**Treatment.**—If seen early the treatment depends upon whether or not there is evidence of arterial failure distal to the trauma and whether hemorrhage forces surgical intervention. If such conditions exist, the treatment should be surgical with exploration of the trauma site after

thorough cleansing *Control of the artery* should be obtained both *proximally* and *distally* before disturbing the injury site

*Removal of Clots* —Clots should be evacuated dead tissue debrided and the artery explored If there is a clot the artery should be opened in its long axis and the clot removed Thrombosis distal to the trauma may be present and this clot likewise should be removed Through the opening the involved artery should be explored until a free flow of blood from above is obtained

The clot may be removed like an embolus by a suction tip or catheter by grasping it with a forceps or by a wire corkscrew This latter is made by winding the end of a large-sized silver wire on a probe and inserting it intraluminally into or through the thrombus A forcible pull usually will loosen the thrombus In some manual milking of the artery has evacuated the thrombus Due care must be taken to prevent its distal passage

It may require ingenuity as in embolectomy to remove the thrombus but the effort should not be abandoned until the thrombus has been removed Venous clotting usually accompanies the arterial occlusion due to stasis If seen early these clots can be removed and the vein ligated and resected proximally to the clots and well away from the trauma site (See Embolectomy Technic pages 442 to 443 )

In all surgical procedures careful cleansing with sulfonated detergents and Hexachlorophene is fundamental Adequate blood for transfusion must be available Chemotherapy is always used It is suggested that the reader consult the chapter on Injuries to Arteries pages 321 to 351

*Sympathetic interruption* should be considered in each case to relieve the attendant spasm This therapy has had a thorough test in the recent war in Korea Sixty five per cent of all war wounds involve the extremities Most of these are attended by spasm which is physiologic in Nature This spasm reduces hemorrhage and squeezes out clots in the main and collateral vessels *After the definitive surgery or where the adequacy of the circulation* remains in doubt sympathetic interruption by blocks or surgery should be instigated If the thrombus is of long standing or if the thrombus was incompletely removed sympathectomy is required Chemical interruption of the sympathetics is of short duration but if no other method is available immediately this should be attempted (See chapter on Sympathectomy page 487 )

*Conservative Management* —Some clinics have followed a conservative course of treatment of arterial thrombosis This regimen included care of the part to prevent injury to it and a mechanical effort to supply blood to the limb by intermittent venous occlusion This therapy has been ineffective in our hands

*Suction Pressure Boot* —The suction pressure boot advocated by Herrman[9] Landis and Hitzrot[10] Theis[14] and others has been used in some clinics This method relies for its efficacy on the negative pressure created by the suction established in a cylinder in which the foot is placed The suction applied is rhythmical and is continuous. These investigators have reported excellent results with this modality The experiences of our Clinic however have not been satisfactory and we have discontinued its use Other clinics also have stopped using this modality

Venous occlusion to help arterial occlusion has always appeared to be in the class of "gadgets" The patients and, unfortunately, some doctors delight in placing a limb in some machine and turning a switch which will "improve or cure" the patient In vascular diseases there is no "push-button" method of getting well

At the meeting of the Society for Vascular Surgery, 1951, Smithwick[15] and his associates presented some proof that venous occlusion as a measure of increasing arterial supply was of no value Their experiments demonstrated that venous occlusion decreased the amount of blood entering the limb With an ingenious set-up, and working on cats, the amount of blood entering the limb with or without venous occlusion was accurately measured. In general there was a definite reduction each time the venous occlusion was applied

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Chapter

# 20

## ARTERIAL ANEURYSMS

### *True and False Aneurysms, Aneurysms of the Aorta Aneurysms of the Peripheral Arteries*

**Definition.**—An arterial aneurysm is a rupture or an abnormal dilatation of an artery which results in a pulsating tumor This mass is composed either of the wall of the artery or contiguous structures or clot but it has a direct connection with the artery If the entire wall is dilated for a certain distance a *fusiform type aneurysm* is created If the sac or wall is made up of the vessel structure itself it is called a *true aneurysm* If the wall is destroyed and the blood is contained by blood clot or by surrounding structures then the aneurysm is a *false aneurysm*

**Anatomy**—Arteries are composed of three layers The *inner coat* is an intact layer and is transparent and colorless It is elastic and consists of a layer of pavement endothelium of which the nuclei are fusiform oval or round This intimal layer has been called tunica intima of Kolliker "[31] Beneath this endothelium is a subendothelial layer which varies in size depending upon the size of the vessel There is a third or elastic layer which is composed mainly of elastic fibers placed in a longitudinal direction and was called by Gray the ' fenestrated membrane This varies again in size depending upon the size of the artery but in such vessels as the aorta it has a considerable thickness The *middle coat* consists principally of plain muscle fibers which are arranged in lamellae circularly placed around the vessel These vary again depending upon the size of the vessel In the very small arteries this coat may have only a single layer but in the larger ones there may be up to four layers The elastic fibers unite to form lamellae in the larger vessels and alternate with layers of muscle fibers This coat is closely connected to the fenestrated membrane of the inner coat In the very large vessels such as the aorta there is a large amount of elastic tissue and some connective tissue in this coat It is this layer which forms the thickness of the artery itself The muscle fiber cells are about 50μ in length and contain rod-shaped nuclei The *external coat* is mainly connective tissue with some elastic fibers in it There is considerable elastic tissue in this layer so much so that Gray described it as a separate elastic layer the tunica elastica externa This layer is the largest in the arteries of medium size In the larger vessels the external coat is thin but in smaller vessels it is relatively thicker As the vessel approaches capillary size this layer thins out, becoming only a membrane and finally disappears. The vessels vary greatly in their thickness depending up-

parently upon how much stress and strain may be encountered For example, in the cranium and the vertebral column, the vessel is extremely thin, the thinness being particularly in the external and middle coats Around these arteries are sheaths which are made of fibro-areolar tissue Between the sheath and the vessel itself is fine areolar tissue and frequently this sheath also encloses the vein and at times the nerves All of the arteries are supplied with blood through small nutrient vessels called the vasa vasorum These arise from a branch of the artery or from another vessel at some distance from the part they supply They penetrate apparently the external coat only although in some large mammals vessels have been traced to the middle coat Fine veins return this blood to the accompanying vein There are also lymphatic vessels and nerves The latter are supplied through the sympathetic system and some through the

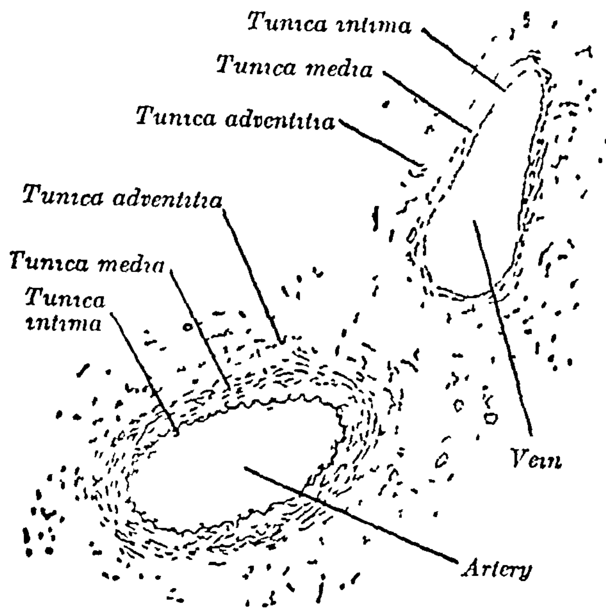



Fig 103 —Section of artery and vein in childhood Various layers and relative size illustrated (Gray's Anatomy )

cerebral spinal nerves They form plexuses on the outside of the larger vessels and run along the smaller arteries in a plexiform manner They supply particularly the muscle tissue of the middle coat, and in this way control contraction and relaxation of this coat Indirectly, therefore, they supply the amount of blood sent to the part

**Embryology** —In development the major vessels seen in the adult do not arise in the embryo They begin as a capillary network and these enlarge until the larger arteries and veins are developed At times the vessels are developed as outgrowths from the enlarged stem. Many of the embryonic vessels disappear or are replaced by other arteries The arteries in their development arise as segments from the primitive dorsal aorta The seventh segmental artery, for example, is of special interest It forms the lower end of the vertebral artery. From this seventh segmental artery the entire left and the greater part of the right subclavian arteries are formed This is the only one of several segmental arteries which

contribute branches to the upper limb bud which persists. The axis artery which is the primary arterial trunk of the lower limb arises from the dorsal route of the umbilical artery The femoral artery originates from the external iliac to form a new channel along the front side of the thigh and to communicate with the axis artery above the knee The axis artery above this communication then disappears except for a small portion of it which remains as the inferior gluteal artery Of the axial artery only two other segments persist One forms part of the popliteal artery and the other the peroneal artery Thus one sees that in its development the circulatory system on both the arterial and venous sides arises from the same common bed The persistence or lack of persistence of certain segments which can be followed if one studies the embryology makes the possibility of congenital defects in these vessels easily understood Further weakness in the closure of certain segments or persistence of embryonic stems on both the artery and vein sides can explain the development later of congenital arterial or arteriovenous aneurysms.

**Etiology** — Any condition which weakens the blood vessel wall or increases the intraluminal pressure may cause an arterial aneurysm The arterial aneurysm may be a dilatation of the arterial wall similar to the blowout of the automobile tire through the weakened casing This is the so-called true aneurysm in which the wall of the tumor thus produced is made up of the dilated and stretched vessel Where the artery has completely ruptured the wall of the sac is no longer made up of the artery itself In this instance the contiguous structures such as muscle fascia bone viscera or even skin may contain the arterial pulsating blood

1 **Congenital Arterial Aneurysms** —Congenital arterial aneurysms are rare The medial coat may be thin undeveloped or absent with subsequent development of aneurysms at any stage Where there is a weakness in the wall either due to a lack of the development of the usual layers or to some other congenital weakness a dilatation may occur quite early in life These aneurysms are found most often intracranially in the anterior part of the circle of Willis or in the internal carotid artery Rupture causes fatal cerebral hemorrhage Arterial aneurysms on a congenital basis are seen usually with other vascular anomalies

2 **Acquired Arterial Aneurysms** —The acquired arterial aneurysms result from two causes They may follow trauma either of a direct or indirect type They may develop also after some disease has weakened the artery With this second type a mild trauma may be a precipitating factor

A *Traumatic Arterial Aneurysms* —These aneurysms occur most often after a laceration of the vessel A gunshot or stab wound an auto or airplane accident or some flying metal or glass often is the source Twenty per cent of the arterial aneurysms operated at our Clinic were caused by trauma During wartime the incidence increases

The laceration of the artery may be complete or incomplete The complete laceration usually results in gangrene The incomplete laceration may not be entirely through the artery wall With arterial pressure the wall may dilate If the laceration is through all of the coats but the artery still is in continuity by part of the wall a pulsating hematoma may develop which will become an arterial aneurysm The clot begins to pulsate as the arterial pressure recovers from spasm and shock. The clot s soft center

permits the arterial blood to circulate within it and soon the blood begins to flow into the distal arterial component The soft clot becomes endothelialized and part of the peripheral artery circulation Its walls thicken and fibrose as the clot is organized. Such a pulsating hematoma becomes an arterial aneurysm

Operative injury of an artery may be a cause for aneurysm Repair of an arterial injury or an operation requiring opening of an artery may result in an aneurysm Every wound, especially gunshot, shrapnel, or stab, close to a major vessel should be suspected of and observed for possible aneurysm development

B *Diseases Causing Arterial Aneurysms.*—Four of every five arterial aneurysms coming to operation are the result of a degenerating disease process In some, trauma was a contributing cause

1 *Syphilitic Aneurysms.*—Untreated syphilis results in aneurysms in between 1 and 2 per cent of the patients Arteritis results from untreated syphilis. It is seen most often in the ascending aorta, the aortic arch, the descending aorta and in the abdominal aorta It occurs rarely in the extremities. The spirochete produces an obstruction of the vasa vasorum secondary to an inflammation of the periaortic lymphatic structures The muscle layers degenerate and aneurysm follows Efforts at repair may delay development of the syphilitic aneurysm These aneurysms are fusiform or saccular in type

2 *Arteriosclerotic Aneurysms*—Arteriosclerosis as a cause for aneurysms is increasing with longevity More patients reach the age in which arteriosclerosis is a predominant factor The medial coats if diseased may rupture. A calcium deposit or plaque is the usual site of rupture The size of the aneurysm depends on the tension in the vessel, the local status of the blood vessel, the contiguous structures and the degree or success of repair. Slight trauma in the presence of arteriosclerosis may precipitate the aneurysm

3 *Mycotic Aneurysms*—The aorta and other arteries may be weakened by a suppurative process such as actinomycosis, tuberculosis or other infectious diseases Embolic arteritis may follow subacute bacterial endocarditis Septicemia, pneumonia and typhoid fever or a local infection around a major vessel may weaken the vessel wall and cause its rupture. These aneurysms occur at sites subject to stress and strain Thus, the femoral, the axillary and the popliteal arteries may be involved Pathogenically, the lesion follows an abscess in the wall involving the vasa vasorum and thus interfering with the blood supply to the part of the vessel involved

4 *Diabetes Mellitus*—The diabetic frequently is subject to arterial aneurysms, due to the arteriosclerosis which accompanies diabetes mellitus in all stages after a certain number of years

5 *Lead Poisoning*—Lead poisoning also may have a weakening effect on the blood vessels and this may be the site of an arterial aneurysm

6 *Gout* and *Tuberculosis.*—These are other debilitating diseases which may be etiologic factors, especially in their later stages

7 *Malignancy*—The invasion of the arterial wall and nearby structures by a malignant growth is a not infrequent cause

8 *Fungus Invasion* —The fungus organisms may cause destruction of the wall and produce an aneurysm

9 *Arteritis* —Periarteritis nodosa temporal arteritis or any of the other types of nonspecific arteritis may weaken the artery by inflammation and ulceration An aneurysmal dilatation may occur at such sites Some of the so-called idiopathic aneurysms may be in this group See Arteritis pages 546 to 552

10 *Burns Roentgen Rays Radium or Radioactive Isotopes* —These also may cause arterial aneurysms

11 *Embolic Aneurysms* —An embolism may enlarge and soften the intimal and medial layers of the site of lodgment with secondary aneurysmal dilatation The increased arterial pressure behind such an obstructive lesion may precipitate the wall break *It is in patients with diseased* vessels and hypertension that such aneurysms develop These occur most often at the bifurcation of the aorta, the internal-external iliac divisions the femoral and femoral profunda junctions or at the bifurcation of the popliteal artery The rupture of a calcium deposit or an atheromatous plaque in a major vessel may weaken the artery and at that point an aneurysm can develop Thus both the site of origin or lodgment of an embolism may be the place where an aneurysm develops

12 *Idiopathic Aneurysm* —In some aneurysms the exact cause is not apparent A congenitally poor blood vessel structure particularly of the muscle may be the cause Many of these aneurysms develop from arteriosclerosis The idiopathic arterial aneurysms should decrease in number with further study

**Aggravating Cause** —A trauma may be the actual activating cause or aggravating force which presents the aneurysm clinically Thus I have seen an aneurysm occur after a very minor trauma to a patient s legs from the slight injury of a car bumper The aneurysm developed in both popliteal spaces within a few days

Medicolegal problems may arise from the questionable etiology of these aneurysms. The relative part of the disease and that of trauma must be considered in each instance. One of our patients developed an aneurysm of the radial artery while lifting some files from a shelf These files were similar to those she lifted each day She was sixty five years of age and had arteriosclerosis The disease caused the aneurysm although it was difficult to rule out the lifting as a possible aggravating cause Many such aneurysms however develop while the patient is in bed

This industrial problem has been discussed under the subject of Cardiovascular Diseases and Trauma (See pages 804 to 814 )

**Symptoms and Diagnosis** —In discussing symptoms and diagnosis the subject will be covered in general and then specifically as to sites of occurrence It is apparent that the symptoms must vary with the vessel involved An aneurysm in the upper extremity may cause slight distal ischemia due to collateral circulation Such an aneurysm will be diagnosed often only on the development of a tumor An aneurysm in the popliteal space may be unnoticed until the attendant claudication brings the patient to the doctor Many aneurysms of the aorta are first diagnosed on a routine

chest x-ray or a flat plate of the abdomen The major symptoms present in the development of aneurysms follow

*Tumor Mass* —One of the earliest symptoms of an arterial aneurysm is a *tumor mass* This mass is caused by the dilatation of the arterial wall or by the false wall The tumor is always present and is a diagnostic sign unless some contiguous structure hides it

At times it can be demonstrated only by certain positions of the limb For example, an aneurysmal dilatation of the femoral or popliteal artery, just as it comes through the adductor canal, may be entirely hidden by the semimembranosis muscle and tendon until flexion of the leg relaxes the

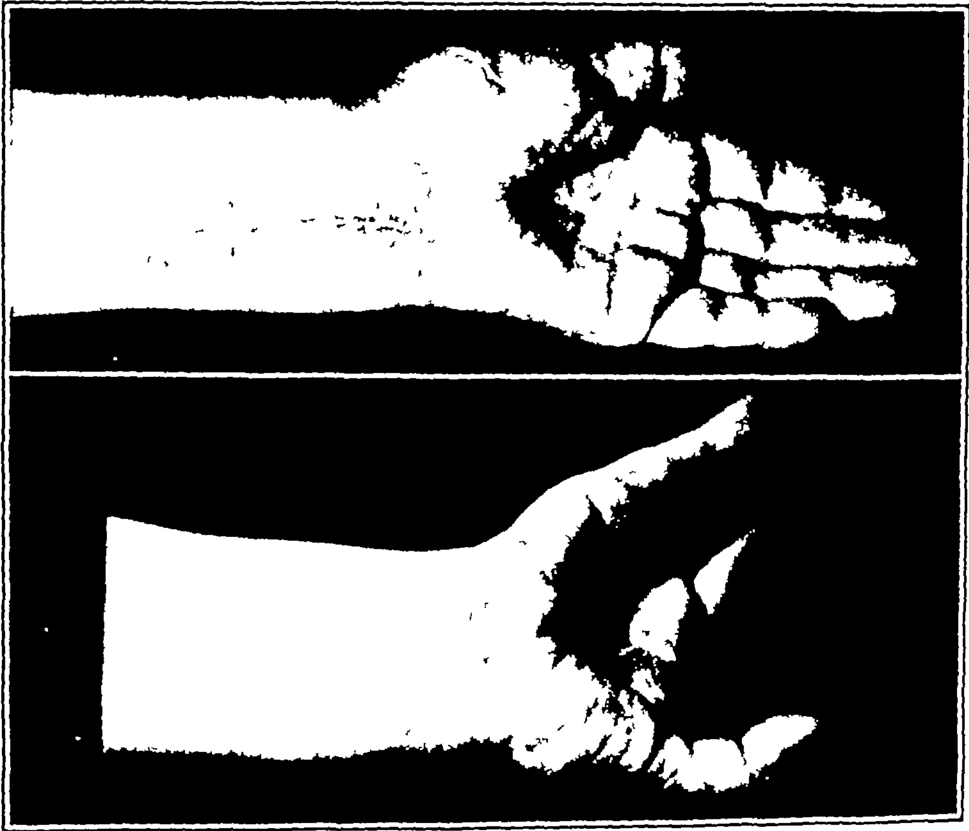


Fig 104 —Aneurysm of the right ulnar and left radial arteries Arteriosclerosis associated with minor trauma was the cause

tendon and permits exposure of the tumor mass An aneurysmal dilatation of the iliac artery was completely hidden for a time by viscera in the overlying peritoneum Many aortic aneurysms are diagnosed accidentally by roentgen ray or only at operation for some other lesion This same masking may occur in the neck or thorax and more often in the cranium In the head, pressure signs may be early In the thorax, roentgen ray or fluoroscopy may be of great help in the diagnosis

The tumor mass usually will be *expansile* The expansile pulsation is synchronous with the systolic heart beat This expansile pulsation is not seen always because at times the external clotting obscures it or the surrounding tissues so obliterate the pulsation that the expansile portion is not palpable

Errors occur in the diagnosis of this tumor mass because other tumors lying contiguous to large vessels may transmit the vessel's pulsations. As the tumor mass increases in size, the pulsation may be obliterated by a clot overlying the central liquid portion of the mass. In some, the wall degenerates and the sac itself consists only of contiguous structures—muscle, fascia and layers of blood clot, the so-called *false aneurysm*.

*Pain* is a common symptom and may be due to pressure of the tumor mass on some sensitive part. Pain also may be due to a lack of blood supply to the part supplied by that artery. Such pain is due to ischemia and with inadequate circulation in the legs results in *claudication*.

The pain of aortic aneurysm is an early and significant one. The erosion of bone occurs soon and thus increases the symptom of pain. Pain causes two out of every three patients to consult the doctor. A missed aneurysm diagnosis most often is due to the failure of the physician to perform a *complete* physical examination. One recent patient had seen two "specialists," neither of whom palpated or listened to an enlargement in the patient's popliteal space, which was an arterial aneurysm. In two patients the initial signs were of *venous* pressure and *occlusion*. In one, pulmonary embolism had occurred from the clot in the popliteal vein. These two patients were hospitalized and treated for their secondary clotting while the underlying cause for the clotting, pressure from a popliteal aneurysm, was undiagnosed. There have been innumerable similar instances and these again point to the dislike of many physicians to perform a *complete* physical examination.

*Pressure signs* develop as the tumor mass gets larger. These signs and symptoms vary with the site of the aneurysm. Thus pressure of the mass may become so great as to force its way to the skin surface. There have been occasions in which there has been a rupture through the skin, an aneurysm necessitans.

*The part involved may increase in size* as the tumor enlarges.

*Variations of Circulation.*—With the large tumor, the *pulse distal* to it is *weaker* than on the other side; in later cases it will be entirely absent.

The *oscillometric readings* and the *blood pressure* are *decreased* on that side. In the *lower extremity*, the involved side has signs of failing circulation with *numbness* and *coldness* of the part. This point emphasizes the importance of determining the pulse, blood pressure and oscillometric readings on all extremities, not just the one closest to the examiner.

In a few, a *distal embolus* may be the original symptom with *acute occlusion*.

*In aneurysms of the ascending aorta*, the variation in the circulation of the two upper extremities may be extreme.

*Bruit.*—A bruit often will occur early in these aneurysms as the blood whirls into the area of weakness or sac. After a time, there is a compensatory clotting and later laminations and fibrosis, which may reduce or eliminate the bruit. The bruit is due to the whirling of the blood in the sac and therefore depends upon the size and the liquid part of that sac. In arterial aneurysms the bruit is synchronous with systole, in contra distinction to the arteriovenous aneurysms, in which there is a to-and-fro murmur.

*Trophic changes* are due to ischemia and disuse of the part. These are distal to the aneurysm and are similar to those seen after arterial occlusion. The nails may atrophy and "buckle" and be the sites of fungus invasion. The digits become shiny, wasting and subject to skin breaks. There may be a decrease or absence of hair.

*Atrophy.*—There may be atrophy in the affected limb due to the lack of adequate circulation below the aneurysm.



FIG. 105.—Gangrene of the foot distal to undiagnosed popliteal artery aneurysm. Patient had been treated for a venous thrombosis which was caused by aneurysm pressure.

*Cerebral Symptoms.*—In aneurysms of the carotid or vertebral vessels or in the brain, the symptoms vary from signs of mild pressure and syncope to hemiplegia and convulsions. These signs and symptoms, therefore, depend entirely on the site of the aneurysm. One of our patients who had an aneurysm of the anterior cerebral artery developed early signs of extreme hunger, eating everything that was placed before him. This is a symptom seen frequently in lesions in the anterior cerebrum. He rapidly developed attacks of syncope, unconsciousness and incontinence and had convulsions before his death from the aneurysm of the cerebral artery.

There are two sources for the blood supply to the brain, the internal carotid arteries and the vertebral arteries. Because of this double cerebral blood supply and the circle of Willis, an aneurysm of one or more of the

branches of the aorta does not necessarily cause cerebral lesions Some irregularity in the circle of Willis however may cause changes The extremely variable symptoms therefore can be based upon any anatomic pathologic basis. The part is subject to sudden changes in the tension of the blood In patients with a sudden mental disturbance or unconsciousness an aneurysm of the circle of Willis should be considered Some of these patients have been treated for a psychosis until their aneurysm ruptures leaving no further doubt as to the diagnosis Since these aneurysms are eradicable their early detection is important

**Pathology** —1 TRAUMATIC ARTERIAL ANEURYSMS —The pathologic picture varies of course with the site and duration of the aneurysm In most cases there is an incomplete laceration of the vessel The wall may balloon out if it is not completely divided If this occurs the aneurysm may be fusiform in shape and its walls are then composed of the weakened vessel wall supported by surrounding tissue Most often the artery wall is entirely divided at least in part A hematoma then is formed For a time there may be no extension of the process the clot acting as a cork. Later the intra-arterial pressure creates a false sac or forms an enlargement of the weakened vessel wall

The walls of the hematoma become reinforced by the surrounding structures which later comprise the walls of the aneurysm There is clotting in these walls followed by fibrosis and lamination In time the wall becomes a thick, leather like sac made up of these laminations and the fascia or the surrounding muscles

There will be inflammation fibrosis and scarring around the sac. In aneurysms of the abdominal aorta or those in the thorax where there is no counter pressure hemorrhage may be alarming and fatal Clotting may occur with the development of a false sac The endothelium soon lines such a sac which may be of a fusiform or saccular type The wall may balloon out with connective tissue proliferation attempting to strengthen the wall An exudative reaction between the sac and the surrounding tissue is common with scarring

On section the wall of the aneurysm may show its own thinned out layers depending on the degree of dilatation There will be an inflammatory reaction around it In the false wall the section will show layers varying from blood internally to more organized clots which become laminated, thickened and replaced by fibroblasts and scar The external coat always presents evidence of inflammation and repair The contiguous structure may be part of the wall and inseparable from it When rupture occurs there will be evidence of laceration of the wall with free blood lining the rupture The wall varies in thickness

Bone may be destroyed by the aneurysm pressure resulting in bone necrosis

2 NONTRAUMATIC ARTERIAL ANEURYSMS —The pathology depends on the disease causing the aneurysm There is usually a ballooning of the wall in the so-called true aneurysm type. This bulging may continue until such time as there is no longer any wall left Rupture then occurs and the surrounding tissues may become the sac. As the condition progresses the clot laminates and at the periphery becomes of the white type being re-

placed by fibrosis as the periphery is approached. In the center, the lumen becomes red and less firm, with a liquid and mobile collection of blood in this area If the condition persists for a considerable time, there is usually an effort toward calcification of the wall, in many of these, the outside has a stone-like appearance

The pathologic picture includes pressure on contiguous organs, sometimes with atrophy of muscles, visceral adhesions, destruction of nerves or bone, and pressure and thrombosis of veins The growth of an aneurysm accelerates when it strikes a resistant structure and it will erode bone With this aneurysmal dilatation, there is a diminution of the blood supply peripheral to this area and therefore atrophy and other evidences of inadequate circulation

In the aneurysms of the arteriosclerotic type, the wall of the vessel may be arteriosclerotic with typical pathologic changes and evidence of fracture Calcium deposits or plaques may be present in the wall, and at times the wall may be entirely replaced by calcium Arteriosclerosis in these cases will be present in other parts of the vessel

The disease which caused the original weakness of the wall will be evident whether it be tuberculosis, gout, lead poisoning, syphilis, or arteriosclerosis In the aneurysm due to syphilis an inflammation occurs in the periaortic lymph system caused by the spirochete This causes an obstruction to the vasa vasorum With this obstruction there is an atrophy and degeneration of the wall of the vessel, particularly of the muscle layer The aneurysm caused by syphilis occurs most often in the ascending and arch of the aorta It also occurs in the descending and abdominal aorta but more rarely in the peripheral arteries It can be diagnosed by the typical pathologic findings of these diseases and their effect on arteries In the sac and adjacent to it will be collateral arterial vessels This fact is important if direct therapy to the aneurysm is contemplated because preservation of such collateral vessels is imperative if the blood supply distally is to continue

**Prognosis.**—The prognosis in any arterial aneurysm depends upon the cause, site, size, and duration of the aneurysm and the possibility of treatment The prognosis is poor in aneurysms of major arteries where the area distal is dependent entirely on this vessel for circulation This includes aneurysms in the thorax, the abdominal aorta, the heart, and the brain

The prognosis of aneurysms caused by trauma is much better than those caused by disease, provided the vessels are not diseased Their therapy will be discussed later

In all aneurysms, especially those which occur in the extremities, there is a marked tendency to develop collateral circulation Sufficient time has elapsed usually for the development of such collateral circulation prior to the time the patient applies for definitive treatment. In many of these cases, the prognosis is excellent if correct surgical therapy is supplied

**Treatment**—The treatment of arterial aneurysms is primarily surgical and should be so considered Small aneurysms occasionally close spontaneously by a deposit of fibrin from the blood within the sac These instances however, are the exceptions to the rule,

Where surgical intervention is contraindicated either due to the position of the lesion or the general condition of the patient medical measures are instituted These are simple and consist fundamentally of maintaining the patient s resistance and reducing the arterial pressure by restricting physical or emotional activity Drugs which tend to lower the arterial pressure may help

*History of Arterial Aneurysm Treatment* —The adage that there is nothing new in medicine and surgery probably is true and nearly every type of treatment has been tried The first effort at controlling arterial aneurysms was by proximal arterial compression The compressing instrument varied from thumbs to types of bandaging The tourniquet as advocated by Morel[29] in 1674 also was used Distal and proximal compression of the artery also was tried but was ineffective

The first attempt at ligation according to Babcock[3] was made by Antyllus in the third century Anel[2] in 1710 ligated an aortic aneurysm placing the ligation directly proximal to the sac John Hunter s classic operation[25] in 1785 consisted of four ligatures which were placed at a distance proximal to the sac constricting but not occluding the lumen

In 1790 Pere Pott[40] reported that the ablest medical men preferred amputation to ligation a mortality figure of 46 3 per cent explains this reasoning

Distal ligation tried by Brasdor[9] and Wardrop[47] failed as did combined proximal and distal ligation of the artery

Halstead s operation[19] consisted of the application of an aluminum band around the vessel to reduce the caliber of the artery This band was applied proximally or distally in the hopes that by reducing this lumen the collateral circulation would be stimulated and clotting in the sac might occur This procedure has been found to be unsound and dangerous Rupture at the band site occurred Efforts to insert a coiled aluminum band or other foreign material in the artery proximal or distal to the aneurysm have not been successful

The introduction of foreign substances within the sac to stimulate coagulation was developed primarily for those aneurysms which were not accessible The earliest work of galvano-puncture or needling by Philetus[36] in 1829 and by Petroquin[37] in 1831 or scarification by MacEwan[26] or needling by Valpeau[31] all failed The various other substances inserted in the sac included watch springs, horse hair iron wire and finally wiring through a fine needle with galvanism by Moore and Corradi[32] in 1879 Babcock[3,4] reported that over 50 per cent of the patients thus treated by wiring died within two months and very few lived a year after such wiring More recently results have improved

*Modern Therapy* —The general surgical principles as applicable to all types of arterial aneurysm will be discussed The specific surgical management of arterial aneurysms in various parts of the body then will be considered in detail

Many arterial aneurysms can be corrected due to the development of better surgical technics antibiotic therapy and the anticoagulant drugs The general surgical technics include the following

(1) *Excision of the Aneurysm with End to End Anastomosis* —This anatomically is the treatment of choice and should be used always where

the continuity of the circulation is necessary for the life of the part. It should also be used whenever the defect caused by the aneurysm excision will permit a bridge to be constructed. Rerouting, stretching and flexion of the part helps in the therapy.

This operation is not difficult if the arteries are not diseased. This method was employed successfully recently for the treatment of an aneurysm of the brachial artery in a boy aged twelve. Collateral circulation was inadequate and the hand would have been sacrificed without the




FIG 106.—A, Radial artery aneurysm in child aged four years. Aneurysm developed two weeks after laceration of wrist. Compression of vessel caused loss of circulation distally. *Operative excision and end to end anastomosis* of radial artery achieved by flexing the wrist.
B, Operation radial aneurysm of A. (Pratt, courtesy of Surg., Gynec. & Obst.)

arterial anastomosis. It has been used on vessels as small as the radial artery in a child aged four.

(2) *Excision of Aneurysm with Repair of the Artery.*—This method has a wide application. In the past when most aneurysms were of luetic origin and the septic problem had to be considered in each artery repair, the suturing of aneurysmal defects in arteries was unsuccessful in most instances. We use it increasingly, and where applicable it solves a difficult problem easily. Nearly all traumatic aneurysms can be repaired. The proximal and distal control of the artery is obtained. New and old clots

are then removed The opening in the artery is visualized by opening the aneurysm sac. Often the arterial opening will be found to be small It is suggested that all aneurysms be opened before they are excised The arterial defect in no way corresponds to the aneurysm size Many difficult bridging operations can be prevented in this way The defect can be sutured with fine arterial silk If necessary this suture line can be reinforced with a layer of the sac Eight per cent of 92 arterial aneurysms have responded to this type of operation

Seven other similar end to end arterial repairs have been successful

Goldyne and Gardner[17] have commented upon the meagerness of the literature on aneurysms of the radial artery Their statement that "the radial artery like the ulnar artery may be ligated anywhere in its course without endangering the viability of the extremity" cannot be accepted especially in children Spasm may play a part but in many instances, the interruption of either artery in the arm can result in gangrene of some of

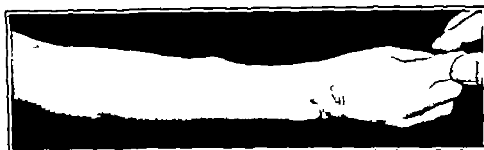


Fig 107 —Traumatic aneurysm of ulna artery boy aged ten. Treatment excision of sac repair of artery with surgical cure.

the digits or the entire hand Since the ulnar artery is the end artery this does not occur as often after ligation of the radial artery In one of our patients age four with an aneurysm of the radial artery, its occlusion produced signs of circulatory failure The age of the patient probably was an important part [43] When an arteriovenous fistula exists, ligation of the artery proximal to it often results in gangrene

(3) *Excision with a Homologous Venous or Analogous Artery Graft* —Th excision of the aneurysm with a vein transplant has been used satisfactoril many times The technic of blood vessel suturing is simpler than that o bowel anastomosis Valuable experience can be gained for this work b practice on the cadaver and on dogs (See Fig 98 p 336 and Fig 9( p 337 )

I have successfully bridged a gap in a common carotid artery with jugular vein transplant where its ligation would have caused a hemiplegia

In both of the above technics, the hydrodynamic law that the wal pressure at any point in a vessel with a circulating media is inversel proportional to the rate of flow applies The rate of flow in an artery i rapid and the wall pressure therefore is low The vein graft permits the us of the patient s own vessel Where an artery graft is used a donor mus be obtained because of the obvious damage to the donor site of using th patient s own artery The donor grafts must be from undiseased youn

donors and reasonably fresh. Grafts have taken after being kept in the bank for fifty days The technic of obtaining, storing and using such grafts is described on pages 340 to 343. The author has inserted one for an aneurysm at the bifurcation of the aorta which included the iliac arteries Twelve per cent of our arterial aneurysms have been treated by venous or arterial grafts The future use of these methods has great potentialities Whether or not the vein dilates and needs support is still under study

(4) *Obliteration of the Aneurysm (Matas[30]) with Muscle Implant*[11] Matas,[30] in 1888, was the first to use the technic of obliteration of the aneurysm This procedure is safe It preserves the collateral circulation and, if performed carefully, it is the operation of choice in most aneurysms where the above technics are not applicable The operative success is

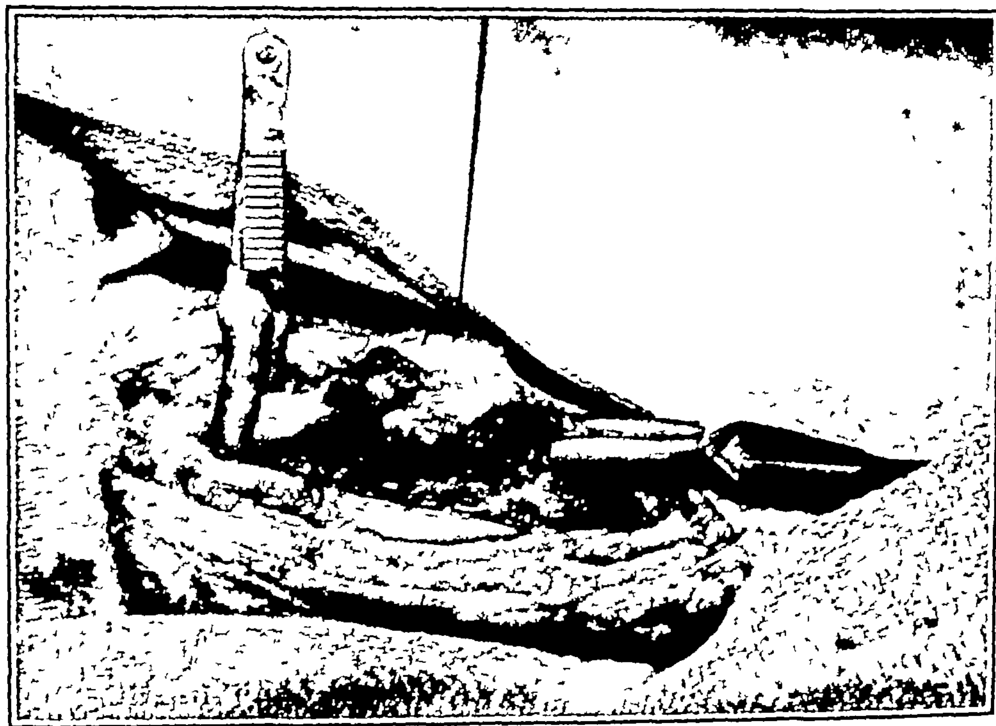


FIG 108 —Ulnar artery aneurysm Dissection, opening of sac, repair of artery Sac used to reinforce suture line (Pratt, courtesy Angiology)

based on the fact that collateral circulation develops around and in the wall of any aneurysm For success of the operation this existent collateral circulation must not be interrupted or traumatized

The operation consists of

(*a*) Control of the circulation proximal and distal to the aneurysm

(*b*) Opening the sac and evacuating all new and old clots, debris, and calcification

(*c*) Closing each collateral opening into the sac from within the sac All sutures should be of silk

(*d*) Ligating the main artery entering and leaving the sac All ligatures again are of silk

(*e*) Retention of the sac Removal of the sac defeats the principle of the operation by interrupting the collateral circulation

(*f*) This procedure was modified in our Clinic by placing a contiguous muscle flap into the sac after closing each collateral opening within the sac and then closing the sac over the muscle core to help obliterate it This muscle implant fills the defect in the sac more adequately than can be done with any form of suture or plication of the sac wall We have performed this operation successfully 42 times with the loss of one patient and the small toe of another It is surgically simple to sew the old sac over this muscle The muscle was divided in the original technic but now the

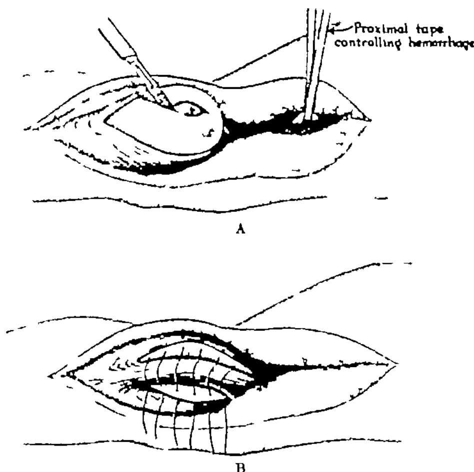



Fig 109 —Aneurysmorrhaphy (Matas type) A Proximal control of artery and incision of sac Laminated clots presenting B Insertion of muscle implant to obliterate the aneurysm sac Any contiguous muscle may be used. No recurrence has followed this technic (Pratt courtesy of Surg Gynec & Obst.)

ends are left attached The results seem to be even better and the blood supply to the implant thus is maintained

(*g*) To prevent recurrence the proximal and distal arteries are sutured and closed from within the sac

(*h*) To this technic we have added opening the accompanying vein removing any clots by thrombectomy and ligating and dividing the vein [a] Matas reported a mortality of 4.5 per cent, with gangrene in only 3.5 per cent and secondary hemorrhage in 1 6 per cent of the cases, and this testifies to the efficacy of this type of operation.

Our mortality of 1 death (2 3 per cent) in 42 operations of this type and no major gangrene, although smaller in number, seems conclusive proof that this operation is the one of choice in most of these large aneurysms if not repairable

*Rupture of Arteriosclerotic Aneurysm* —Aneurysms due to arteriosclerosis may remain stationary for months or years Some trauma may cause the rupture of the wall There may be severe hemorrhage due to dissection by the blood Shock may ensue The treatment of such an aneurysm depends upon its site, size and the patient's status Shock should be treated first If the circulation is adequate, definitive therapy of the aneurysm



Fig 110 —Calcified aneurysm of left femoral artery after gunshot wound Direct trauma to knee ruptured aneurysm Treatment. evacuation new and old clot, obliteration and muscle implant Lumbar sympathectomy also performed

should be delayed If the circulation is failing, operation will be required as soon as the shock has been counteracted Control should be obtained proximally and distally to the aneurysm The treatment usually is an obliterative procedure unless a repair or graft is possible In the aorta, wiring is most often effective

(5) *Perianeurysmal Irritation* – It has been known that if the aneurysm could be made to occlude slowly enough, collateral circulation would develop in sufficient quantities to nourish the parts distal to the aneurysm Nature has clotted even aneurysms of the aorta on occasion Perianeurysmal irritation is an effort in this direction. In the manufacture of one form of polythene, a substance called diacetyl phosphate develops This substance when placed with a polythene covering around the aorta of puppies gradually occludes such vessels in one year The work of Poppe,

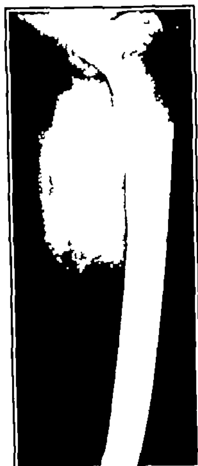


Fig 111 — X ray aneurysm of femoral artery Calcified wall ruptured due to trauma with hemorrhage. Original cause of aneurysm bullet wound ten years before Note bullet superimposed on femur Dissection of the fascia

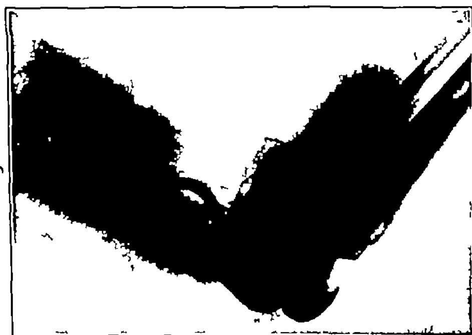


Fig 112 —Arteriogram of popliteal aneurysm. Large size of sac shown with small liquid center Note collateral circulation shown in the sac wall, emphasizing the importance of *not* removing the sac which would destroy the collateral circulation.

De Olivera,[39] and Pearse[35] led to the use of this substance in the treatment of aneurysms. Where other therapy is impossible, or where the aneurysm is at an inaccessible site or the patient's condition precludes any more active therapy, these aneurysms can be covered with the polythene. A fibroblastic proliferation then develops. The polythene which is used must have the diacetyl phosphate on it. It is manufactured by DuPont and has the term Polythene type NV-7-14.

A nonirritating cellophane (300 PUT-71) should surround the irritative substance to protect the contiguous structures from the reaction. The polythene should not be tied in place as this produces an irresistible band against which the aneurysm will force itself until it ruptures. We have used this type of therapy in 8 per cent of our arterial aneurysms.

(6) *Intrasaccular Wiring.*—This method which originated with the work of Corradi and Moore[33] was revived by Blakemore.[7] It is discussed in detail on page 392.

(7) *Combined Wiring and Wrapping with Polythene.*—These two methods have been used jointly. We have felt that wrapping in combination with intrasaccular wiring is best effected by steel mesh.

(8) *Intrasaccular Wiring and Steel Mesh Cloth Wrapping.*—Steel mesh is now made in all sizes and strength. It is an alloy and noncorrosive. Wrapping large aneurysms with it has been combined with intrasaccular wiring, and seems particularly effective in large abdominal aortic aneurysms. The steel mesh incorporates itself in the wall of the aneurysm and thus a stronger wall is formed to contain the wire and the expected clot. We have used this in 6 patients with massive aneurysms and 4 of them are alive, the oldest being over two years.

(9) *Artery-Vein Anastomosis Distal to Aneurysm (Babcock).*[3,4]

*Other Methods. Foreign Body Tube.*—The use of a foreign body tube with or without a vein lining has been attempted for forty-five years, the first modern reports of success accompanying direct blood transfusion attempts. More recently, this tube technic has been revived, using tantalum and inserting a vein for an endothelial lining. Rupture has occurred in most cases at the tube end. In a few, this method may be lifesaving temporarily. The proximal ligation operations rarely have any place in the therapy. All but one of our reported abdominal aorta aneurysms which were ligated died of perforation. In that one instance a fascial pack was placed proximal to the tie. The use of slingshot rubber, polythene ligations, fascial ties and reinforcements, etc., are not considered successful.

**Preoperative Preparation.**—The usual preparations for any surgical operation should be augumented in aneurysm surgery by provision of the following:

(1) *Available* blood, with pressure apparatus for rapid arterial and venous transfusions
(2) Sterile tourniquets
(3) A sterile stethoscope
(4) Experienced assistants
(5) Sufficient hemostats
(6) Tapes and rubber bands
(7) Suction
(8) Special clamps such as Pott's, etc.

Preliminary preparation includes postural exercise tests for adequacy of collateral circulation and at times temporary constriction of the vessel to stimulate the collateral circulation

In most cases sympathectomy should precede or immediately follow the operation If this is impossible sympathetic blocks should be performed immediately

## HEAD AND NECK ARTERIAL ANEURYSMS

Arterial aneurysms in the head and neck should be surgically treated after allowing a sufficient time for the collateral circulation to develop If the condition is progressing too rapidly one must operate early The involved artery should be explored control obtained proximally and if possible distally, and the amount and condition of the collateral circulation inspected [6]

The treatment of choice is *excision* and *end to end anastomosis* This is possible in most traumatic aneurysms in the head and neck With the availability of the anticoagulant drugs more of these operations can be performed

If anastomosis is impossible and the circulation appears adequate the artery should be obliterated without interfering with the collateral circulation At times pressure on the artery proximal to the aneurysm will determine whether or not this will be successful Other technics for arterial aneurysm detailed on pages 372 to 378 have application at times

**Aneurysm of the Common, Internal and External Carotid Arteries** — Conflicting literature has been accumulated on the results of ligation of the common and internal carotid artery Nearly all textbooks describe cerebral changes in fairly high percentages after ligation of either the common or internal carotid artery Thus Babcock[3,4] states that ligation of the common carotid is followed in 26 per cent by marked cerebral changes of which 50 per cent are fatal his figures being derived from the reports of Halstead [18] Thorek[40] states that cerebral softening coma and death occurs in 20 per cent of common carotid ligations In general the older books state that there is a mortality rate of from 20 to 50 per cent Wounds of the common carotid artery have a high mortality rate Cerebral complications occurred in 50 per cent with 55 per cent of these fatal In 17 wounds of the common *carotid artery there was 47 per cent fatality and in another* series of 25 carotid artery wounds 44 per cent died Many neurosurgeons consider the internal carotid artery ligation much more serious than the common carotid Thus Schorstein[47] believes emphatically that the ligation of the internal carotid artery to be much more dangerous than the common carotid Olivecrona[34] reported 2 patients in whom ligatures of the internal carotid artery caused neurological signs whereas common carotid ligature was well tolerated in both Dandy [14] Sweet and Bennet [49] and others believe that if there are no neurologic changes in ten to thirty minutes either vessel can be safely ligated The possibility of blood flow from the external to the internal carotid has been suggested by some 20 authors Rogers[46] ligated the common carotid artery in 19 patients with no complications Locke[31] reported 33 cures in 34 patients ligating the internal

carotid for intracranial aneurysm Thus one can prove either side of this question statistically In aneurysms which have been present for some time, collateral circulation is probably adequate through the circle of Willis It is the author's practice to prepare the patient with pressure over the aneurysmal site for many weeks prior to operation, as suggested by Matas The vessel involved is then obliterated by a tape for a time (forty-five minutes to one hour), and if cerebral symptoms do not manifest themselves, the vessel is ligated or obliterated In only one instance have there been cerebral signs.

*Case Reports* —A patient, aged forty-seven, with arterial aneurysm of the internal carotid artery had this vessel excised No cerebral signs appeared for twenty-four hours, at which time a hemiplegia and progressive coma were followed by signs of cerebral change on the opposite side Death ensued in forty-eight hours The patient was a known hypertensive for twenty years Postmortem examination fortunately was obtained The sections through the brain showed an egg-sized area of hemorrhage of the left side and a walnut-sized hemorrhage on the opposite side This patient had concomitant cerebral hemorrhages, which unfortunately occurred at the time of his operation for aneurysm The operation was not responsible for it other than to increase his emotional tension.

A similar occurrence is also relevant and to the point The patient who was to have an operation for arteriovenous fistula on a Monday developed a hemiplegia the night before. Had this hemiplegia been delayed for twenty-four hours it would have been considered the result of the operation Since cerebral vascular accidents occur frequently in patients with such disease, this possibility should be kept in mind The patient should be watched for any signs of cerebral anemia or beginning hemiplegia

In other aneurysms, especially those of traumatic origin, the artery may be freshened and anastomosed end to end

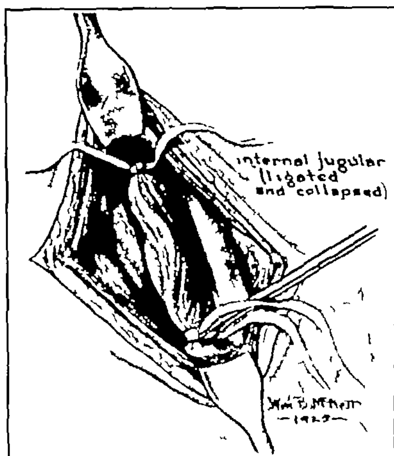
In certain cases, *venous grafts* can be used to bridge the gap This has been done successfully in one instance in the carotid artery

The treatment with *endothelium-lined tubes*, such as those applied by Murray[335] and others, has been used at times and may be satisfactory. A tendency to necrosis at the point at which the ligature holding the tube in the vessel has been applied should be remembered Fatal hemorrhage has occurred with rupture of the vessel at such a point of ligation

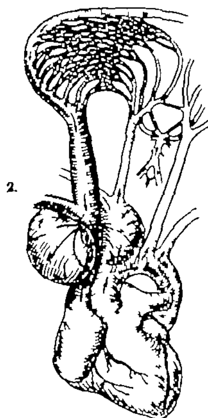
*Artery Grafts* —See pages 340 to 342 The use of homologous artery grafts has been discussed

## THORACIC AORTA ANEURYSMS

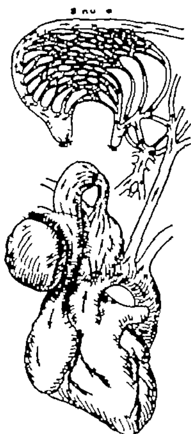
1 **Resection and End to End Anastomosis.**—In the smaller aneurysms of the thoracic aorta, *a resection* and *end to end anastomosis* is the treatment of choice; this applies particularly to those aneurysms of traumatic origin[41,42] Such an operation is comparable to the one for coarctation Collateral circulation may be as intense and the dissection as difficult as in the operation for coarctation This factor depends upon the length of time the aneurysm has been present, the age of the individual and the condition of the aorta When the artery is markedly diseased, such an approach will not be feasible In such cases, other measures are indicated




1



2.



3

FIG 113 —Babcock carotid-jugular anastomosis for aneurysm of the ascending aorta. 1 Anastomosis proximal end of the carotid artery to the cardiac end of the jugular vein 2 Diagram showing peripheral circulation with increased aortic and aneurysmal pressure. 3 Reduced peripheral resistance and intra-aneurysmal pressure by the anastomosis. (Courtesy Dr W W Babcock and Surg Clin. N A.)

2 **Excision Operation with Repair of the Aorta.**—This operation has an application in aneurysms of the thoracic aorta It is advisable to open each such aneurysm before excising it and trying to replace the vessel with a graft The aneurysmal sac may overlay the aorta and the aortic opening may be very small Some surgeons have excised large sacs and then, to their consternation, found that the aortic opening could have been repaired simply This is true particularly in traumatic aneurysms, but may be true in congenital aneurysms or those due to disease, especially syphilis

3 **Homologous Vein or Analogous Artery Grafts.**—Such grafts also have a place in the thoracic aorta, especially if the aneurysm is on the descending part of the vessel Where the aneurysm arises at a point where necessary collateral branches come off, the replacement may be impossible The use of grafts in diseased arteries has some application The diseased arteries are difficult to suture and the graft emplacement may fail for technical reasons beyond the surgeon's control

4 **Obliteration of the Aneurysm.**—Obliteration may be possible in some aneurysms of the thoracic aorta This applies also to aneurysms of the innominate artery or of the first portion of the subclavian artery The technic of obliteration of a large sac requires control proximally and distally The sac then is opened and each collateral opening within is individually sutured In some patients the control of hemorrhage is difficult since each opening may spout blood as from a spigot Proximal and distal control at such a time mav not be sufficient to stop the bleeding Many surgeons have a difficult time in understanding this factor In such cases the blood coming out of these collateral openings originates above the control tapes With this bleeding the surgeon should remember that the best hemostatic agents are his fingers An assistant's digits can be used as plugs in these openings One by one these should be sutured A technical point of help is to get one suture through the edge, above and below the opening The tension on these sutures will help control the bleeding and bring the opening into the field for sewing *Adequate blood* must be present and running By adequate blood is meant 4000 cc and this must be available in the operating room and not in the blood bank After each opening has been closed off, the main entrance and exit openings can be closed by intraluminal sutures Care should be exercised not to interrupt the collateral circulation running in the sac and in contiguous structures To help obliterate the sac, a section of muscle can be incorporated The sac is then obliterated by mattress sutures of silk over the muscle, again placing them so as not to injure the vessels running in the sac

Operations on these aneurysms are formidable The patient, the family and the surgeon must accept the risk The danger of hemorrhage always will be present When the emergency arises, the surgeon must avoid panic in himself or in his team He must be quick and often ingenious Pressure or constriction with the hand will control most hemorrhage This control should be maintained until the patient's status is satisfactory for definitive action The surgeon must use God's hemostats—the hands and fingers

5 **Periancurysmal Irritation.**—This method has been used in thoracic aneurysms with some success It is discussed on pages 378 to 379

6 **Intrasaccular Wiring.**—Intrasaccular wiring as a treatment for aneurysms is based upon the fact that Nature "cures" an aneurysm occasionally

by clotting it The original work was done by Corradi and Moore[8] with iron wire. Blakemore[7] revived the method using a #34 gauge, B & S insulated coin silver wire This wire permitted an introduction of a coagulation current In recent years we have used #32 alloy steel wire without coagulation The varying thickness and the different degrees of degeneration in the aneurysm have made us fear that heating the wire might destroy part of the wall The aneurysm is exposed under direct vision Previous technics for aneurysm wiring by blind needling were instituted due to the great danger of opening the chest in the days of poor anesthesia We believe greater danger is created by such a technic today No efforts are made to dissect the aneurysm free The needle is one with a stylet which can be removed, and a second needle with a fish mouthed end on it is introduced The wire passes through both needles The wire is fed in manually to the center of the sac After some of the wire has passed it can then be introduced by the inner needle This fish mouth is roughened and carries the wire in and then can be slid back on the wire to carry in a new section

The wire is rolled on two spools from either end so that at the center there is a doubled piece of wire which starts the feeding Thus no sharp ends are introduced and each segment of wire inserted is doubled With experience one can hold back one end of the doubled piece slightly while pushing forward the other—thus making a loop inside to reach the greatest circumference of the sac In general 200 to 600 feet of wire can be introduced dependent upon the size of the sac and the technic of introduction

The great advantage of the wiring technic is that the pain associated with the aneurysm usually is alleviated The pain is due to the thrust of the blood column within the sac which causes pressure on nerves and other sensitive structures and even erodes bone The wiring reduces or eliminates the thrust Patients who have been on opiates no longer require them The length of life seems to be prolonged If the patient lives no longer than he would have otherwise he at least lives happier and without pain Rupture of the aneurysm usually occurs in months or years depending upon its size the vascular status and the success of the therapy Often the rupture occurs at a site other than the area wired In larg aneurysms we have introduced the wire from both sides by two needles the assistant inserting wire at the same time as the surgeon

7 **Combined Intrasaccular Wiring and Wrapping with Polythene** —Thi method has been used in some patients

8 **Intrasaccular Wiring Combined with Steel Mesh Cloth Wrapping** — This has been the most effective measure in our hands for the enormous o 'hopeless' aneurysm of the aorta

9 **Foreign Body Tubes** —Foreign body tubes have been used to perform an anastomosis. The shortcomings of a foreign body in a pulsating vesse have been discussed on pages 345 to 347 In an emergency such a tube ma continue the circulation temporarily

10 **Babcock s Operation**—*End to End Anastomosis of the Artery an Vein Distal to the Aneurysm* [1,4]—After some experimental work, Babcock' operation was evolved

Babcock's operation is based on the hydrodynamic law that the pressure on the walls of a tube containing a moving liquid progressively decreases as the velocity of the liquid is increased In an aneurysmal dilatation therefore, if one could increase the rate of flow in the liquid passing through the sac and decrease the peripheral resistance against which this fluid must be forced, this would allow the pressure at any point in that vessel, particularly in the sac of the aneurysm arising from it, to be decreased.

That this is successful has been shown by the shrinkage in the size of aneurysms after such an operation

An end to end arteriovenous anastomosis is different than a lateral anastomosis such as occurs traumatically In the latter, a parasitic circulation is developed and the heart must hypertrophy In an end to end anastomosis however, the blood returns to the heart more rapidly The rate of flow in this system is increased and the wall pressure decreases as the rate of flow rises together with the elimination of peripheral resistance

*Technic* —For the ascending aortic aneurysm the carotid artery and jugular veins are anastomosed Under procaine infiltration anesthesia, an 8 cm transverse incision is made 2 cm above the right clavicle The platysma myoides and the sternocleidomastoid muscles are divided The carotid artery and jugular vein are mobilized Hemorrhage is controlled by small rubber tapes The upper ends of the common carotid artery and jugular vein are ligated and transfixed with #0 black silk Five mattress type 4-0 silk sutures are then placed in an everting style to unite the proximal end of the carotid artery and the cardiac end of the jugular vein in an end to end manner The anastomosis is completed by a running everting suture of 4-0 silk The tape on the cardiac end of the jugular vein is opened first The carotid artery is then released Any bleeding point is sutured The wound is closed in layers The danger of cerebral ischemia exists in such an operation This operation has been performed six times for thoracic aneurysms

## ABDOMINAL AORTA ANEURYSMS

Aneurysm of the abdominal aorta is one of the most difficult lesions with which surgeons are confronted, and surgical cure is difficult Statistically, aneurysm of the abdominal aorta is reported ten times less frequently than aneurysm of the thoracic aorta, but these figures were compiled when syphilis was the main cause In our experience, the abdominal aorta is affected as often as the thoracic aorta when arteriosclerosis is the cause of the aneurysm Since abdominal aortic aneurysms are somewhat of a separate entity, they will be treated individually as to cause, signs and therapy

The saccular type of aortic aneurysm (due to syphilis) lends itself well to excision and arterial suture, the aorta being restored to normal size The possibility of recurrence exists Eight patients were reported on by Bahnson[55] In 6, the sac was excised and the aorta sutured. This lends impetus to this type of operation

**Etiology.**—In the past, syphilis was the cause of nearly all aortic aneurysms With better control of this disease, the syphilitic aneurysm more rarely is seen Most of these aneurysms today are the result of arterio-

sclerosis In a study made by Estes[16] of 102 abdominal aortic aneurysms admitted to the Mayo Clinic prior to 1948 97 were found to be of arteriosclerotic origin These aneurysms were considered to be arteriosclerotic if there was x ray evidence of calcification in the aorta, in other arteries, or the diagnosis of arteriosclerosis had been made clinically The diagnosis of syphilis was entertained only if there was a positive serological test or sufficient clinical evidence in the absence of arteriosclerosis Both of these lesions were considered as a cause of the disease if the above criteria were present Trauma was a minimal factor although at times it may have been a precipitating one

These figures are in line with our findings and are in marked contrast to the previous ones of Kampmeier[28] (57 per cent syphilis) Scott[43] (74 per cent syphilis) Hubeny and Polack[21] (75 per cent syphilis) and Pratt-Thomas[14] (15 out of 17 patients syphilitic) The increase in longevity (95 per cent of Estes patients were over fifty and they averaged sixty five years of age) and the better control of syphilis are the causes for the variation of etiology Approximately 70 per cent died of rupture In 10 per cent syphilis occurs concomitantly Five of every 6 patients are over fifty years of age Hypertension or trauma may play a part in the development of abdominal aorta aneurysms by loosening or weakening some arteriosclerotic plaque In a few an aortitis of mycotic etiology causes sufficient weakening of the vessel to permit the aneurysm to develop Diabetes may be a cause as all diabetics have arteriosclerosis

**Symptoms** —In about one-half of the patients with abdominal aorta aneurysms there may be no early symptoms The most common symptom is pain which is not localized and may be abdominal or be referred to the back due to the pressure A tumor mass occurs in all patients This may be masked by certain signs of an acute abdomen This develops in 1 to 2 per cent with some bleeding There have been many cases in which the first signs were those of pressure In others increasing constipation is noted In the later stages melena and the vomiting of blood from rupture of the aneurysm into the colon or duodenum are not uncommon There may be repeated small hemorrhages before the fatal termination due to massive rupture of the aneurysm Intestinal obstruction is a complication

**Diagnosis** —Aneurysms of the abdominal aorta and its branches give confusing signs at times and are diagnosed later than other aneurysms Early signs are frequently those of a tumor mass sometimes diagnosed without the previous knowledge of the individual or the physician on a routine roentgen ray examination The diagnosis is often made by the surgeon during a laparotomy The shadow of the tumor mass with displacement of other viscera may be suspicious In many cases the calcification in the wall is discernible At times the earliest symptoms are those of obstruction of the duodenum or colon the tumor mass being secondarily discovered Hemorrhage may cause signs of an acute abdomen

Changes of posture may be necessary to bring this mass into diagnosable view either physically or by roentgen ray The mass may have an expansile pulsation which is synchronous with cardiac systole In other instances so much clotting has occurred that the pulsation is merely transmitted or it may be masked by the overhanging viscera If the

patient is placed partially on his right side so that all viscera tend to slide away from the left side of the abdomen, the mass can be palpated more readily In this position, too, the bruit, which can be heard in average cases in only approximately 1 in 8, can be heard in over half the patients

In a few, aortography has been necessary to make the diagnosis The aortogram is a somewhat formidable procedure in aneurysm of the abdominal aorta due to the usual attendant arteriosclerosis

**Pathology.**—The aneurysm in the abdominal aorta tends to dissect distally and may involve either iliac artery and extend far down one or both of these vessels Its proximal progress is usually limited by the renal arteries, although in certain instances, these become involved secondarily, causing symptoms of renal insufficiency There is an inflammatory reaction surrounding these aneurysms which may cause marked adhesions and angulation of the bowel

The dissecting type of aneurysm usually stretches the medial layer. There is a tendency of the aneurysm to push everything before it This tendency increases where resistance is met Erosion of the vertebrae is not uncommon

The pathologic changes in the vessel walls vary with the type of disease causing the aneurysm In the arteriosclerotic type, typical arteriosclerotic changes occur with vessel wall destruction. Where the aneurysm is of the fusiform type, all of the vessel walls are present but are dilated. In many, the wall is missing entirely, with the surrounding tissues forming the aneurysm sac

**Prognosis.**—The length of life from the time an abdominal aorta aneurysm is discovered clinically on the average is nine months, and the length of life after such a patient is admitted to the hospital is one month The prognosis therefore is extremely poor The surgeon is justified in taking somewhat heroic measures in an effort to prolong the life of such individuals

One recent report summarized 454 cases, and the average length of time from the diagnosis of the abdominal aneurysm to the death of the patient was one year. Kampmeier's[27] report of 68 abdominal aorta aneurysms in the Charity Hospital in New Orleans showed that one-half, or 38, died a month after admission and that all were dead in eight months. The cause of all but three deaths was rupture of the aneurysm

**Treatment**—For many years, the treatment of aneurysms of the abdominal aorta consisted of bed rest and sedation Early reports described attempts to clot the sac Moore, and later Corradi,[33] tried to do this by the insertion of iron wire The use of a spring wire or a fascial plug to cause intravascular clotting or coagulation also failed Halstead's[18] and Crile's[13] metal bands designed for partial obliteration also were unsuccessful

*Modern Therapy*—While the therapy varies greatly with the cause and extent of the aneurysm, the following technics today generally are used in the surgical management of aneurysms of the abdominal aorta:

(1) *Excision of the Aneurysm with End to End Anastomosis*—If the aneurysm is small, especially in those of the traumatic type, an excision of the aneurysm with end to end anastomosis may be tried This technic is much the same as in the end to end anastomosis of the aorta in coarctation of the aorta (See pages 89, and 382 )

(2) *Excision of Aneurysm and Repair of Aorta* —This operation probably can be used only in the traumatic aneurysms In very small aneurysms due to other causes this technic might be applied In the abdominal aortic aneurysms due to arteriosclerosis however, the aortic wall is destroyed and no repair is possible Syphilitic aneurysms can be excised

(3) *Excision of Aneurysm with Homologous Vein or Analogous Aortic Graft* —The use of these grafts at this stage is limited Veins appear to contain aortic blood pressure at least for a time In the animal experimentation some dilatation occurs in the vena cava grafts at the end of a year This work is still experimental An aorta from the vessel bank has been sutured into the human aorta successfully on several occasions Four times a graft of the abdominal aorta including the iliac arteries has been



Fig 114 —Rupture aneurysm into duodenum. Point of rupture of aneurysm. Note clotting of sac (Courtesy Dr Joseph E. J King)

inserted The case history of the patient in whom this procedure was performed by the author is given.

*Case History* —American male white age sixty five with signs and symptoms of a pulsating tumor mass of three months duration X ray diagnosis of an abdominal aortic aneurysm was confirmed clinically Operation was performed under spinal anesthesia The tumor mass arose directly below the renal arteries and involved both iliac arteries for a distance of 2 inches A graft was obtained from the vessel bank run by the New York Society for Cardiovascular Surgery Hemostasis was controlled by clamps and the use of rubber catheters The abdominal aneurysm was incised The clots and debris were removed The multiple collateral openings were sutured At this stage the proximal clamp gave way with a tremendous hemorrhage This was controlled by the operator s hand and then maintained by occluding the catheter The graft was sutured in

place with great difficulty due to the disease and friability of the aorta and due to the disparity in the size of the recipient aorta and donor graft The distal ends of the grafts were sutured without difficulty into the iliac arteries At the termination of the procedure, aortic blood was passing through the graft satisfactorily, the feet were warm, and the color good At the end of forty-eight hours, failing circulation in the lower extremities was noted The patient died after seventy-two hours

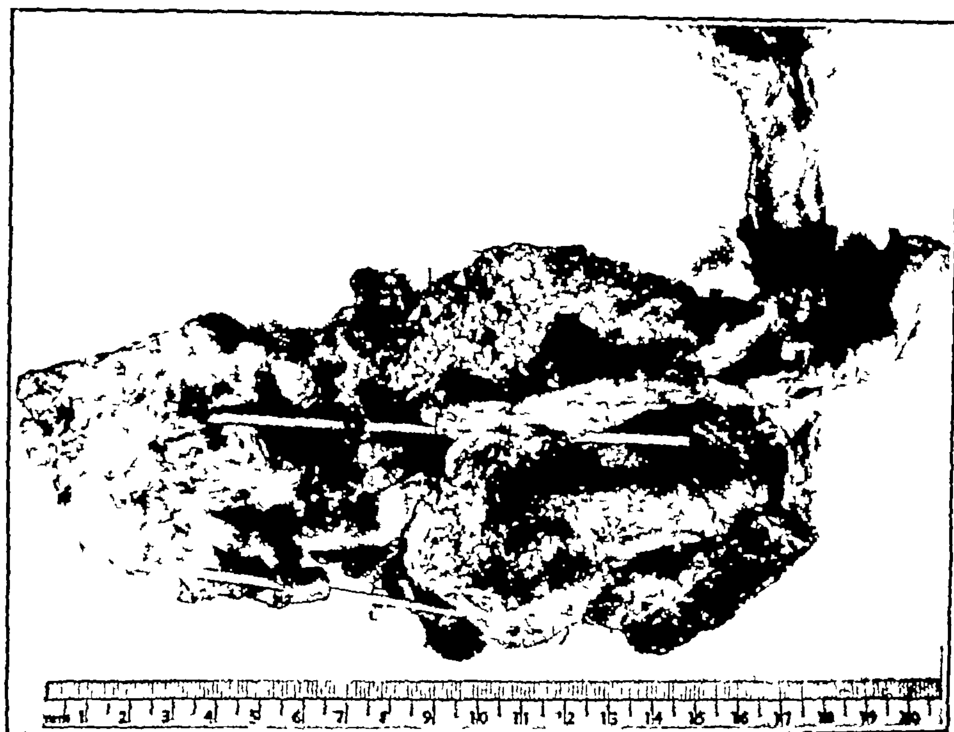


FIG 115 —Aneurysm of the abdominal aorta Treatment by inlay an alogous aortic graft Postmortem specimen Central probe in dilated aorta Inlay graft had been attached by interrupted sutures Iliac arteries of graft attached to iliac arteries of host Disproportion between host and graft aorta was 3 6 cm to 1 5 cm in diameter Patient died fourth postoperative day due to clot in the space caused by disproportion of the host and donor aorta Circulation had been re-established

Autopsy showed that thrombosis had occurred at the site of anastomosis in the aorta The recipient aorta measured 3 6 cm in diameter The donor graft was 1 5 cm The disparity had been corrected by invaginating the larger aorta, and it was at these areas that clot formed The proximity to the renal arteries had precluded dissection to a more normal-sized aorta

(4) *Obliteration of the Sac by Suture and Muscle Implant* — In certain instances, where the aneurysm is small, the obliterative type of procedure can be done satisfactorily, again provided that the dilatation is distal to the origin of such important vessels as the superior mesenteric artery It has been proven in the operating room that obliteration of the inferior mesenteric vessel is feasible in aortic aneurysms without interference to the circulation to the part of the bowel normally supplied by this vessel The superior mesentery and the renal arteries however cannot be interrupted without irreparable damage

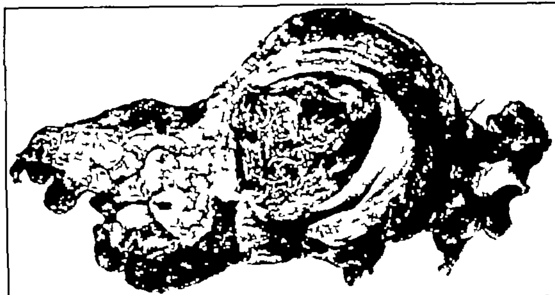


Fig. 116.—Autopsy specimen abdominal aorta aneurysm treated by intrasaccular wiring and extrasaccular steel wire mesh. Aneurysm had ruptured at point of its greatest size. Secondary rupture eight weeks later at point 4 inches proximal to previous site.

Fig. 117.—Obliteration of abdominal aortic aneurysm. Collateral circulation within sac ligated. Proximal and distal openings sutured. Sac packed with muscle and retained.

(5) *Perianeurysmal Irritation*.—The irritative factors inherent in the commercial development of cellophane and its by-products has been the subject of considerable study. The fact that allergy to these substances develops led to investigative work designed to utilize this response to tissue stimulation. The work of Pearse, Page, Harrison, and Harper developed the irritative qualities of the substance.[22,23,34a,35,36] Poppe and de Oliveria demonstrated for the first time the different reactions to the various types of cellophane.[39] Thus, moisture-resistant cellophane and types 300 PUT-71 and 300 T T 62 caused little if any reaction. Polythene-brand of cellophane definitely causes irritative reaction.

It has been found that 1.5 mil polythene (NV-7-14) will cause a marked irritative hyperplastic and eventually constrictive and scarring reaction when applied to an artery.[1] The time necessary for this process to reach its conclusion, at least experimentally, is one year.

This method may be combined with intrasaccular wiring. The polythene must not constrict the aneurysm and this precludes an encircling suture to hold it in place. Such occlusion will result in rupture.

(6) *Intrasaccular Wiring*.—This subject has been discussed under the general therapy and also under Thoracic Aortic Aneurysms. It has its best application in the large aneurysms of the abdominal aorta.

*Technic*.—Under spinal anesthesia the abdomen is opened over the protruding mass. All viscera is packed in the upper abdomen. The retroperitoneum is opened. The aneurysm is wired with #32 steel wire as described under thoracic aortic aneurysms. If there is any bleeding, it is controlled with pressure at the wiring site. A piece of gel foam or oxycel may control any oozing. The retroperitoneum is closed with a continuous suture of 00 plain catgut. The abdomen is closed with chromic #1 for the peritoneum and #32 steel wire for the fascia. Number 35 steel wire is used for skin closure. The appendix is removed routinely, since one such patient developed an acute appendicitis after opening the abdomen for wiring.

(7) *Combined Wiring and Wrapping with Polythene*.—Cooper[10] and his associates performed an excellent investigative study on the aortas of dogs. They combined a partial occlusion by a tantalum band with Polythene cellophane. They were thus attempting to overcome the inevitable rupture at the site of obstruction which had harrassed Halstead,[20] Matas,[31] etc., by producing a fibroplastic exudate at the site of the obliteration. This was successful in most of their tested dogs. While the pathology in the arteriosclerotic aneurysms is far from similar to the healthy aorta of a puppy, there still exists some evidence that this method is worthy of consideration.[8] The author has combined wiring with polythene, encasing as much of the sac as is feasible in polythene. Since the insulated coin silver wire (#34 B & S) is not available, we have used ordinary #30 and #32 alloy stainless steel wire with good results. In some patients with aneurysms of the advanced type, where there is a "bad apple" necrosis and beginning rupture appearance, indeed electrocoagulation may be contraindicated.

(8) *Intrasaccular Wiring Combined with Steel Mesh Cloth*.—This method has been discussed under Thoracic Aortic Aneurysms. The steel cloth is held in place by the retroperitoneum if one is able to find a line of cleavage between the retroperitoneum and the tumor. This is the method of choice in aneurysms which cannot be repaired.

(9) *Foreign Body Tubes* —Foreign body tubes do not seem to have a place in the treatment of abdominal aortic aneurysms Some plastic may be developed

(10) *Babcock Operation* —The principle of the Babcock operation as outlined under Thoracic Aortic Aneurysms has been applied by both Babcock and the author The external iliac artery is anastomosed end to end to the proximal end of the divided iliac vein This increases the rate of flow distal to the aneurysm and this factor plus the reduced peripheral resistance drops the pressure within the aneurysmal sac.

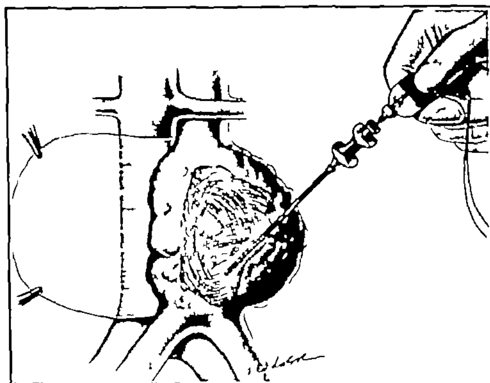


FIG 118.—Drawing of combined intrasaccular wiring and extrasaccular application of steel mesh cloth. As much as 600 feet of steel wire has been inserted in such aneurysms. Mesh tacked externally but does not entirely surround the sac

(11) *Proximal Obliteration* —The first report of a ligation of the aorta was made by Sir Astley Cooper in 1817 [11] Thirty-six subsequent reports followed in which 8 patients survived at least one year Of these the vessel was ligated with cotton tape in 5 fascia lata in 21 and cellophane in 2 The list of complications bespeaks the dangers of the procedure. Erosion, hemorrhage recurrence and intestinal obstruction were causes of death Aluminum silver bands were used by both Halstead[10] and Matas[11] with the idea of gradual or partial obliteration Rupture was usual under the bands Both of these observers believed that this was due to atrophy of the vessels It seems likely however that this occurred because of the constant propulsion of blood against a resistant body the band From time to time this method is tried because Nature herself has shown us that a clot can cure an aneurysm If this method is used an hour-glass type of constriction with the use of cotton tapes or fascia sutures is advocated (See page 396 )

Proximal obliteration has been tried many times All but one of our patients, whose abdominal aorta was proximally ligated, ruptured at the site of ligation The sole exception occurred where a fascial plug was placed proximal to the occluding tie

This problem cannot be studied under an identical pathologic pattern in experimental animals, because an abdominal aneurysm similar to that



Fig. 119 —Abdominal aortic aneurysm, with wiring Loops of wire in large sac Note enormous size of sac (Six hundred feet of #32 steel wire introduced )

in man cannot be produced in dogs The development of this type of aneurysm, with the collateral circulation which necessarily follows, can occur only when the pathologic state that caused the aneurysm in man is present

The solution to this problem, if it is evolved, will follow surgical trials on man himself Treatment of this group of aneurysms with wiring alone has not proven an effective measure, and many times complications have developed That the problem eventually may be solved surgically seems definite By solution of such a problem is meant the prolongation of life



Fig 120 —Pathological specimen of ruptured aneurysm of the aorta the rupture occurring into the thorax. This patient had had an operation for fusiform aneurysm with wrapping of cellophane held in place loosely by a silk suture. Rupture occurred at the site of ligature

Fig 121 —Calcified abdominal aneurysm.

without death from the aneurysm *per se* Any disease which produces pathology as advanced as an abdominal aorta aneurysm must necessarily show itself in a short time elsewhere From the surgical standpoint alone, we can consider the treatment a cure if we can prevent death from rupture of the aneurysm itself. Certain conclusions can be drawn

(1) Abdominal aneurysm is an extremely hazardous condition and will end fatally unless something surgically can be done This applies particularly to the arteriosclerotic type

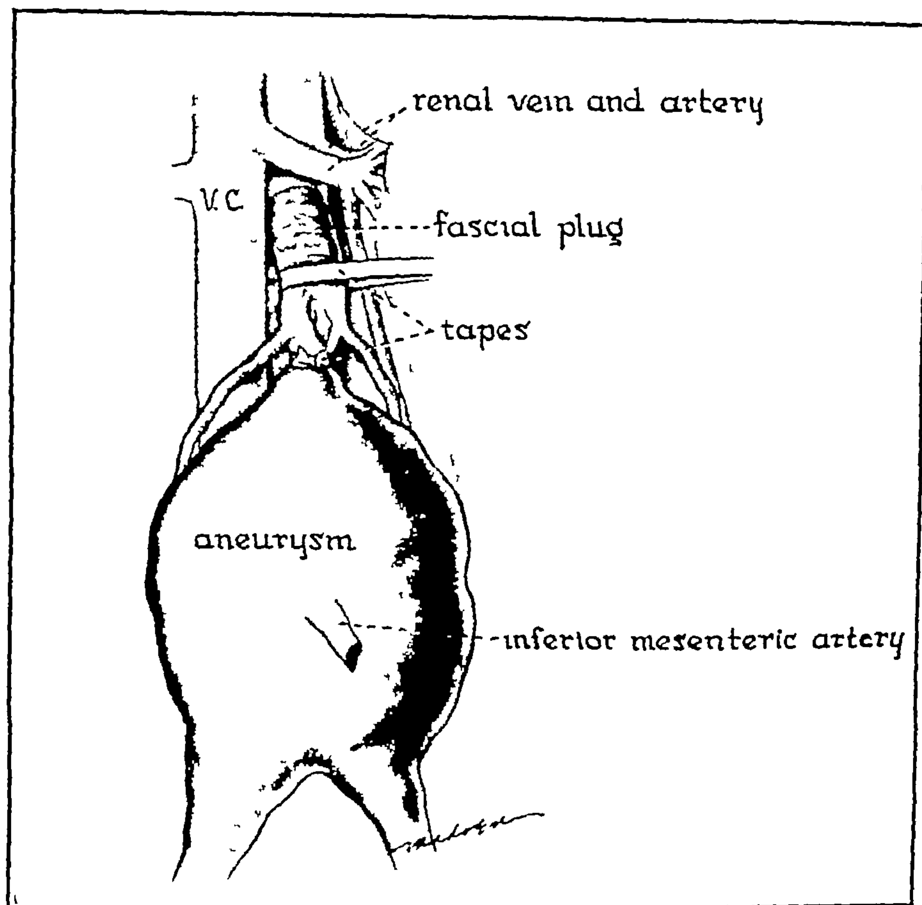



Fig 122 —Surgical treatment of abdominal aneurysm In this instance the aorta was opened above the aneurysm site and a fascial plug introduced proximal to two tapes, the fascia protruding between these tapes to anchor it The tapes were then tied The idea was for the plug to absorb the force of the obliteration of the aortic flow rather than to have it at the actual site of ligation

(2) Obliteration of the aneurysm by clot, if it can be performed slowly, is curative.

(3) A proximal ligation which completely obliterates the lumen of the aneurysm will occlude the aorta, clot the aneurysm and therefore cure the aneurysm If the hazard of the ligation itself and the likelihood of rupture at the point can be overcome, cures could be expected [2,3]

(4) The inferior mesenteric artery can be obliterated with sufficient collateral circulation from the superior mesenteric to avoid gangrene. The essential renal arteries cannot be obliterated in a like manner

(5) A combination of methods of treatment is probably the method of choice in the light of our present knowledge. Intrasaccular wiring combined with extrasaccular envelopment with an irritating substance such as polythene cellophane or steel mesh cloth seems to be the best method of therapy at the present time provided a reconstructive operation is not possible.

(6) An aorta bridge from a cadaver is still in the experimental stage. The bridge supplied by the graft is temporary and the end result is an endothelialization of it and replacement of the graft by the host. (See page 340.)

(7) Resection of the aneurysm if saccular (syphilitic) is feasible and has been successful.[14]

## ANEURYSMS OF THE EXTREMITIES

Aneurysms of the extremities are treated surgically in much the same way as aneurysms elsewhere. The treatment is detailed on pages 388 to 397. The treatment of choice of course is to re-establish the continuity of the

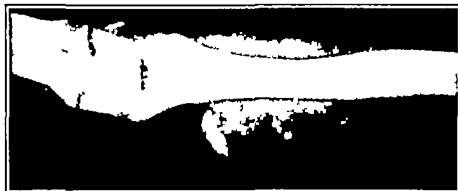


FIG. 123.—Aneurysm of the brachial artery. A small tumor remained in the arm for twenty years after gunshot wound of arm. Hand pressure activated the aneurysm and it increased rapidly in size. Roentgen ray appearance of calcified wall with liquid blood center. The calcium settled to the bottom of the sac. Calcium wall likely broken by pressure. Treatment—surgical obliteration with muscle implant. (Pratt, courtesy of Surg. Gynec. & Obst.)

artery. This is accomplished by end to end anastomosis, repair of the artery, a graft of the analogous vein type or homologous artery type. If none of these methods is applicable the treatment of choice is the obliterative aneurysmorrhaphy of Matas. This operation is facilitated, we believe, by the incorporation of a muscle implant into the sac. The other methods such as perianeurysmal irritation, intrasaccular wiring or reinforcing by fascia, wire mesh, etc. are of a secondary type value and should be employed when the other methods fail or are not feasible. An obliterative operation remains the single best method applicable to the largest number of peripheral arterial aneurysms. In a series of 51 peripheral aneurysms treated by the obliterative method there has been 1 death. This patient died three months after the operation from pulmonary embolism. There has been no gangrene in any of the other extremities with the exception of 1 patient who lost a little toe.

In recent years some surgeons have reverted to extirpation or excision of a peripheral aneurysm. This was the method used prior to the classic work of Matas, and if it is successful at all, it proved the patient had developed an *excellent* collateral circulation. To perform this excision operation one necessarily must interfere with the collateral circulation in the sac and surrounding tissues. This destroys the Matas principle upon which the first successful operation on peripheral aneurysm was developed. Granting that gangrene does not follow in the individual case, if the aneurysm had existed long enough and the surgeon uses sufficient care to protect the secondary circulation, the method cannot be advocated or recommended.

Percentage of arterial aneurysms from our Clinic treated by several methods.

TABLE 31.—METHODS OF TREATING ARTERIAL ANEURYSMS

1. Excision with end to end anastomosis. Treatment of choice. Applicable 10%. Purpose: Restore artery.
2. Excision aneurysm with repair of artery. Applicable 8%. Purpose: Repair artery.
3. Excision with analogous vein or homologous artery graft. Applicable in young individuals—15%. Purpose: Replace artery.
4. Obliteration of aneurysm sac, preserving collaterals (Matas). With muscle implant—37%. Purpose: To eliminate aneurysm without loss of distal blood supply.
5. Perianeurysmal irritation. Polythene (less than 1.0% dicetyl phosphate, XV-7-14). Protect by cover of cellophane 300 PUT—71. Purpose: Gradual obliteration aneurysm while collaterals develop—8%.
6. Intrasaccular wiring. Purpose: Clotting followed by fibrosis—7%.
7. Combination wiring and wrapping with polythene—2%.
8. Peripheral end of artery anastomosis proximal end of vein. (Babcock). Purpose: Remove peripheral resistance and increase rate of flow distal to aneurysms—7%.
9. Proximal obliteration. Complete—obsolete, leads to rupture. Partial—soft rubber—polythene, tantalum, etc., unsatisfactory in humans—7%.
10. Wrapping with steel mesh cloth. Purpose: Reinforce clot.
11. Combination of wiring and wrapping with steel mesh cloth—2%. Purpose: Intrasaccular clotting and fibrosis with sac reinforcement.

TABLE 32.—SUMMARY OF ARTERIAL ANEURYSMS SURGICALLY TREATED
89 OPERATIONS ON 82 PATIENTS

| | *Etiology* | *Site* | *Main Symptoms* | *Prognosis* | *Treatment* |
|---|---|---|---|---|---|
| 1 | Trauma—20% | Any vessel | Mass, systolic bruit, pain, ischemia | Good with operation | See above |
| 2 | Degenerating disease—80% (Arteriosclerosis, syphilis, etc.) | Any vessel, mainly aorta | Same and x-ray calcification | Poor. Therapy eases symptoms, prolongs life | Same as Traumatic type, but usually obliteration |

**Complications of Aneurysm Operation on the Extremity** —The *immediate complications* are (1) hemorrhage (2) arterial thrombosis (3) pulmonary embolism (4) arterial embolism and (5) infection A deficient blood supply may be a later *complication* following an aneurysm operation on the extremity When a patient has had an operation in which the circulation has been obliterated he must be treated for a time thereafter as one would treat a patient with advanced occlusive disease The patient must be made to keep within the limits of the circulation which remains This is important particularly in walking as the collateral circulation may not be sufficient to carry the load required by extensive muscular activity As the patient walks he increases the demand for the blood pumped into his extremities as much as three times and if this blood is not available the use of these muscles must be reduced



Fig 124 —Specimen of large popliteal aneurysm. Patient had been admitted for operation on this aneurysm but later refused Arterial occlusion soon occurred and amputation was necessary Such aneurysms cannot heal spontaneously

*Claudication* will result from overuse of the part Many times it is necessary to keep these patients on crutches for a period of four to six weeks gradually increasing the amount of activity until their limb has shown that it can carry the load required

Other signs of ischemia may occur These include *infections* which will not heal *trophic changes* in the digits *chilblains* or *frostbite* upon exposure to cold

**Postoperative Treatment after Aneurysm Operation of the Extremity** — The treatment of the *inadequate circulation changes* occurring following aneurysm operation of the extremity include

(1) Permanent abstinence from nicotine or other spasm producing drugs

(2) Good hygiene and avoidance of injury and skin breaks

(3) Stimulation of collateral circulation by Sitz baths postural exercises graded exercises alcohol aspirin papaverine hydrochloride oranixon etc.

(4) Interruption of the sympathetic system (see pages 487 to 524)

The surgeon s experience dictates to some extent the operative success in aneurysm surgery as no two cases are exactly alike The altered physiol

ogy and pathology which occur when there is an aneurysmal dilatation in one case may be entirely different from those in another Our results of 266 operations on 197 patients with various types of aneurysms are summarized[42 43]

TABLE 33 —SITES OF 138 ARTERIAL ANEURYSMS

| Site | No. | Site | No. |
|---|---|---|---|
| Aorta | 26 | Radial | 7 |
| Innominate | 4 | Ulnar | 9 |
| Subclavian | 9 | Iliac | 5 |
| Carotid and branches | 21 | Femoral | 12 |
| Axillary | 9 | Popliteal | 21 |
| Brachial | 7 | Tibial | 7 |
| Dorsalis pedis | 1 | | |

TABLE 34 —TWO HUNDRED AND SIXTY-SIX OPERATIONS ON PATIENTS WITH ARTERIAL AND ARTERIOVENOUS ANEURYSMS

| Operation | No. |
|---|---|
| Excision (nearly all AV aneurysms) | 163 |
| Obliteration with muscle implant | 34 |
| Wiring and wrapping | 18 |
| Anastomoses | 14 |
| Wiring alone | 3 |
| Arterial repair | 15 |
| Wrapping with irritant | 7 |
| Ligation alone (four aorta) | 5 |
| Amputation | 4 |
| Others (gastroenterostomy-decompression, etc ) | 3 |
| (Figures exclude sympathectomy) | |
| Total | 266 |

Our over-all operative mortality in the surgical treatment of 138 arterial aneurysms is 9 per cent Of these deaths, all but one were due to aortic aneurysms The single exception died of a cerebral hemorrhage associated with his hypertension Replacement of the vessel by some type of tissue as a graft is the best future hope

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Chapter

# 21

## ARTERIOVENOUS FISTULAS AND ANEURYSMS

*Congenital Arteriovenous Aneurysms Traumatic Arteriovenous Fistulas Arteriovenous Fistulas Due to Disease*

**Definition.**—By arteriovenous fistula anastomosis or aneurysm is meant an abnormal connection between the artery and the vein This may be a direct connection such as follows a wound or the vessels may communicate by means of a connecting sac The connections may be single or multiple

Arteriovenous fistula or aneurysm may be of two kinds—acquired or congenital The etiology symptoms and specific treatment of each type will be described in this chapter

The incidence of arteriovenous fistula is probably much greater than the reports would indicate A few traumatic fistulas close and some of the congenital ones disappear with growth The connection may be achieved by arteries and veins opening into contiguous structures After direct trauma a clot may arise from injury of both the arteries and veins The propulsion of the arterial blood will cause the arterial blood to circulate in such a clot before continuing its progress to the part distal to the injury One part of the arterial blood will return through the venous system without reaching the periphery The rest of the blood continues to the capillary bed The condition may come on after a disease has weakened the vessel walls with breakdown of the arterial and venous walls A rise in the arterial pressure (hypertension) may be a factor It may be also of *congenital* origin This congenital origin is frequent and at times has been misdiagnosed as varicose veins

**Anatomy and Embryology**—In their development arteries and veins are similar Often they arise from the same vascular bud The anatomy and embryology of the *arterial* components are detailed on pages 363 to 365 The *venous* system develops as visceral and parietal divisions The *visceral* consists of two vitelline veins which run in front and later on either side of the intestinal canal These unite ventrally and have anastomotic branches. The superior mesenteric vein opens into the dorsal branch These veins thus form venous rings around the intestinal tract Above the upper ring a plexus of sinusoids develop with the vessels draining the blood to this plexus and becoming the branch of the portal vein. The vessels draining the plexus into the sinus venosus become the hepatic veins These two vitelline veins thus bring the blood from the yolk-sac. The umbilical vein returns the blood from the placenta The *right umbilical* vein disappears but the left one enlarges and opens into the upper venous

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ring of the vitelline veins Later a direct branch develops from the left and right hepatic veins called the ductus venosus The blood then returns from the placenta directly to the heart without going through the liver The *parietal* veins begin as transverse veins (ducts of Cuvier) which open into the sinus venosus An ascending and descending vein opens into each of these The descending vein, a primitive jugular vein, returns blood from the head The ascending vein receives blood from the iliac and hypogastric veins The right cardinal vein persists as the azygos vein The inferior vena cava is complicated in its development but eventually receives the blood from the hepatic veins, the renal veins, the spermatic or ovarian veins and the proximal part of the ductus venosus The Cuvierian ducts are called the right and left superior venae cavae The left one atrophies The development of the venous drainage of the dura and the coronary sinus is not significant to us in this chapter

The veins begin at the venous end of the capillaries as plexuses These join into trunks and enlarge by receiving branches as they course toward the heart The veins are much more numerous and larger than the arteries The walls are thin, they collapse when empty, they freely intercommunicate and have valves which direct the flow of blood toward the heart The superficial veins are in the layers of the fascia but communicate with the deep ones by perforators The deep veins accompany the arteries, usually in the same sheath The larger arteries, *i e*, axillary, subclavian, popliteal and femoral, have one vein The smaller arteries, *i e*, radial and ulnar, have paired veins on either side called the venae comitantes The common embryological and anatomical development makes the formation of arteriovenous fistulas and aneurysms possible both congenitally and due to trauma and disease

**Etiology.**—*Acquired Type*—Arteriovenous fistula was first described by William Hunter in 1757 [34] The cases of acquired arteriovenous fistula seen in the past most often were caused by gunshot and stab wounds Modern war increases this incidence With more rapid and distant travel, serious automobile and airplane accidents occur and many arteriovenous fistulas now arise from these sources Industrial injuries also cause the fistulas As would be expected, such arteriovenous fistulas are most often seen near the areas of large joints, such as the elbow, the hip, and particularly in the popliteal space, which is subject to injury by the bumper of a modern automobile In diseased vessels, the precipitating injury may be slight

*Traumatic Type*—Gunshot or stab wounds are the most common causes for arteriovenous fistulas Flying missiles or parts of a machine are also common causes. The incidence of arteriovenous fistula following extremity injuries in warfare is very high, and in one group of 600 war injuries personally followed, of which 60 per cent were in the extremities, six arteriovenous fistulas were seen [53]

A direct connection between the artery and adjacent vein is not unusual An aneurysmal sac develops between the two if the condition exists for any time There may be several sacs of arterial and venous origin, or there may be an aneurysmal dilation of just the artery or vein Sometimes the artery and vein appear to enter a sac at a similar site In such a connection there are separate arterial and venous connections Rarely are there only

two four or six openings in such an aneurysmal sac The collateral circulation around and into the sac makes the vessels involved innumerable and the surgical dissection therefore is extensive, tedious and necessitates skilled assistants and a great deal of blood replacement The diagnosis in arteriovenous fistula should be suspected

1 On the history
2 If enlarged veins are present which are not otherwise explained
3 If chronic venous stasis with ulcers develops after an injury
4 When a limb is larger warmer or longer than its member
5 When a patient or the physician is aware of a murmur or thrill.
6 If the heart is enlarged without other organic cause for the hypertrophy

Contributory diagnostic help is obtained by oxygen saturation tests arteriography the so-called Branham sign of bradycardia on pressure over the fistula, increased venous pressure increased blood volume increased cardiac output and ischemia distal to the fistula

Arteriovenous aneurysms may occur secondary to certain diseases

1 *Malignancy* —The blood vessel walls may be weakened by malignant changes in the adjacent tissues so as to cause an arteriovenous fistula Direct malignant invasion of the vessel wall with a fistulous development also may occur In sarcomas at times a sudden enlargement of the part with pulsation is not infrequent due to rupture of a blood vessel If there is a venous component in the destructive process caused by the new growth an arteriovenous fistula will be the result

2 *Other Diseases* —Any disease which by its inflammatory reaction its destruction of tissue or by its invasiveness and extension involves directly or secondarily an artery and vein wall or walls may cause a rupture of the artery and vein and therefore a fistula

*Mycotic Infection* —Mycotic disease may also weaken the wall of an artery or vein causing a fistula to occur

*Congenital Type* —Congenital arteriovenous aneurysm is the result of a persistence of an embryonic connection between the arteries and veins Developmentally connections between arteries and veins are common but obliteration normally occurs just before birth, An arrest in such obliteration results in these communications persisting or being inadequately closed In the former case there are direct endothelium lined pathways between the arteries and the veins Such a failure usually is not *localized and there may be multiple or innumerable fistulas These may* occur in any part of the body but are frequent in the upper extremities In the lower extremities many times such fistulas are diagnosed and treated for a time erroneously as varicose veins.

Some vascular discoloration or abnormality often is noticed at an early age The physician consulted usually has advised that nothing be done as the patient might grow out of it

In some patients the obliteration between the artery and vein is a thin one, easily broken by some exercise, trauma or increased intra-arterial pressure

*Another form of* arteriovenous connection is the so-called aneurysmal varix in which an artery is connected with an enlarged varicose vein In others there are many such connections the so-called arterial varices

This is much more common than has been accepted and is the cause of many of the technical problems arising in the treatment of dilated veins These complications are the result of failure to recognize the true nature of the lesion The acute development of arteriovenous connections in the extremities of patients which have been normal previously was described by the author[55,56] as well as by Wright[69] This lesion has been reported more recently by Abdalla[1a] in South America These lesions were precipitated by exercise alone in many instances In some, trauma may have been a contributing factor, but in others, only unusual utilization of the part without additional trauma was the causative agent "The marvel is not that congenital abnormal communications between the arteries and veins occur, but that they do not occur more often in view of the common bed of development of each side of the vascular tree and the enormous constructive as well as destructive changes necessary before the final pattern is reached, that is, the formation of definite arterial and venous channels "[67] A perusal of the literature shows no other reports than those by Pratt[55,56] and Wright[70] on the precipitation of arteriovenous aneurysms by exercise alone This cause should be emphasized as it is not unusual.

Some of the causes for the aneurysms which we have encountered in the last several years and for which we have operated are summarized in Table 35

TABLE 35 —ETIOLOGY OF 126 ARTERIOVENOUS ANEURYSMS

| *Trauma* | *Congenital* | *Degenerating Diseases* |
|---|---|---|
| 30 | 89 | 7 |
| (Including war wounds) | (Some precipitated by trauma) | |

Of 322 operations on 261 patients with arterial or arteriovenous aneurysms performed by the author, 82 per cent were of the arteriovenous type

**Symptoms of Acquired Arteriovenous Fistulas.**—Following a gunshot or b wound, the symptoms may be masked for a time by the surrounding norrhage There is usually a *tumor* which early becomes expansile l pulsates synchronously with the heart beat The part becomes *warmer* h the increased arterial blood flow As the venous connections become ated, the part is *duskier* than the other extremity This cyanosis may st throughout the extremity but is most *pronounced* near the site of the ula The involved *veins dilate* rapidly and in a short time the collateral inches likewise markedly dilate These veins sometimes are treated oneously as varicose veins

*Thrill and Bruit* —A *thrill* will be felt along the involved vessels, which l be greatest at the site of the abnormal connection

A *bruit* will likewise follow the course of the vessels and will be loudest at connection

Both the thrill and the bruit vary with the size and site of the anastosis and usually, but not always, are continuous The bruit is more rked during systole At times, a to-and-fro murmur of equal amplitude heard The bruit in arteriovenous aneurysm thus differs from the bruit arterial aneurysm which is present in systole only

The bruit is caused by the whirling of the blood. It occurs in a large vein or in the sac connecting the artery and vein as the arterial blood with its greater force enters an area in which the pressure is lower. The blood then whirls before continuing its course, this revolving causing the bruit and the thrill. It is apparent, therefore, that in some arteriovenous connections there will be no bruit because the size of the artery entering the sac or vein is small and the comparatively larger size of the sac or the vein is sufficient to take up the arterial blood as it enters without the whirling.

If the diameter of the vessel is decreased by constriction or pressure, as occurs in certain positions, the sac or opening may be compressed and the whirling prevented and the bruit temporarily abated. If clotting occurs in this area of junction the same may be true. In extreme elevation of the part the bruit may be absent. Other positions or postures similarly may



FIG 125.—Arteriovenous aneurysm in the ear of a baby after delivery by forceps. Hematoma at the site of forceps application organized and began to pulsate. Operation two years later with excision of mass and plastic closure. Arterial side of aneurysm was from the postauricular and temporal arteries.

change the degree of the thrill and bruit. If there is a variation in the thrill and bruit at two examinations the patient's position may explain it. Many patients with obvious arteriovenous connections have been mis-diagnosed because no bruits were heard.

With the increased vascularity, the extremity *hypertrophies*. The sweat glands become more prominent and increase in number.

If one applies pressure proximal to the arteriovenous connection the pulse will be slowed; if the pressure is continued the thrill and the bruit may be closed off.

*Pain* usually is a symptom of acquired arteriovenous fistula and may be due to two causes. It may be caused by pressure of the tumor mass on a sensitive part such as a nerve. Secondarily there may be pain with the development of circulatory failure and inadequate blood supply to the muscle.

*Increased heat* always is present with acquired arteriovenous fistula The heat can be felt with the hand and may be an early symptom With a potentiometer, the skin temperature may register from 3 to 12 degrees higher than the adjoining areas

*Claudication* will occur if a large vessel is involved The pain results from deprivation of the normal blood flow to the part

*Other Symptoms* —Other symptoms of arteriovenous fistula depend on which vessel is involved When the fistula is in the carotid or vertebral vessels, cerebral signs due to anemia or pressure may be seen with aphonia, syncope, paralysis, and even hemiplegia

*Atrophy with Arteriovenous Fistula* —In a large arteriovenous fistula, especially of traumatic origin, so much of the blood may return through the parasitic fistulous tract that insufficient blood continues in the normal arterial channels to nourish the periphery As a result there is a deprivation of the normal blood supply to the part Atrophy, loss of tissue and function, and even gangrene may follow. The heart will try to compensate for this loss with increased rate of flow and there may be a larger collateral supply developed

**Symptoms of Congenital Arteriovenous Fistulas.**—Congenital arteriovenous fistulas are caused by some failure of an endothelial closure to occur between the arterial and venous systems The connections usually are multiple and, in our experience, have been observed more frequently on the lateral side of the extremities than on the medial and more often in the upper than in the lower extremities The same symptoms may be present in congenital arteriovenous fistulas as in the acquired type, depending on the size and the number of openings

There usually are innumerable dilated veins fed by small arteries

The thrills and the bruits may be multiple, at times inconstant or absent, and dependent on the size and site of the abnormal connections In those patients with abnormal birthmarks, port wine discolorations, cavernous and simple hemangiomas where the limb increases in size, its temperature is raised and the hair and perspiration are excessive, one should suspect an arteriovenous fistula Bruits and thrills are not necessarily present inasmuch as the connections between the arteries and veins may be small In such instances the larger vein can take up arterial pulsation coming through the small opening, thus eliminating the whirling necessary to cause a thrill or bruit Pressure on the artery above the site of the fistula may or may not cause the bradycardia associated with an acquired arteriovenous fistula In general, the blood is always a brighter color than venous blood The oxygen saturation test will show an increase in the percentage of oxygen over that of venous blood At times, arteriography is necessary to make the final diagnosis

*Distribution and Malignant-like Tendencies* —Congenital arteriovenous anastomoses simulate blood vessel tumors and may be found in any part of the body For example, these arteriovenous connections have invaded the spinal canal like new growth, causing complete paralysis of one limb and extending later to constrict the nerve supply to the other limb These same tumors invade the thorax One such patient died from a massive hemothorax after the rupture of the vascular mass

The presence of the arteriovenous connections in the brain may give rise to the most bizarre symptoms and physical findings Their presence in end arteries such as in the foot may result in circulatory failure in the part They are seen very frequently in the ulnar artery and vein Their branches and collateral circulation may be so great in these instances that surgical extirpation may require complete excision of the circulation to that part The physiopathology of an arteriovenous fistula in the digits varies from that in other parts of the body The fistula diverts the blood to the veins and gangrene may result The arterial blood necessary to reach the end arteries and arterioles by passes these small vessels to enter the low pressure recipient veins Multiple collateral by passes develop and the circulation following the line of least resistance takes the low pressure venous return route rather than the resistant peripheral arterial and capillary bed pathway Amputation in such cases may show so many vessels that control may be difficult or even impossible The vessels grow into the bone It has been necessary to pack the haversian canal with tendons to control the bleeding

The progression and invasiveness of a congenital arteriovenous fistula is not unlike new growth

In one patient an arteriovenous fistula invaded the brachial plexus and its extirpation was impossible without dividing parts of the brachial plexus We have seen such lesions so invade other vital tissues as to make their complete surgical removal impossible

At operation time the blood vessels are found to be extremely delicate distended friable and many times consist of only a single layer of endothelial cells Slight pressure or tension on such a blood vessel causes it to tear and rupture Control thereafter is extremely difficult because of the fragile wall and the vessel s widespread distribution Hemorrhage is severe and the weight of the hemostat often will cause it to pull out with further blood loss

*Cancer-like Tendency* —The invasion by these blood vessels into muscle and other tissues makes their extirpation also difficult They will frequently surround such important structures as a duct from a salivary gland the nerve to the eye the spinal cord lung or other vital parts of the body These congenital arteriovenous lesions therefore are the malignancy of the blood vessel system They will not metastasize and cause death as cancer does but by their direct extension and opening of new anastomoses around vital structures may make complete surgical excision impossible. Eventually they may cause the patient s death by hemorrhage or pressure or both

*Cyanosis* may be most extensive *Pressure gradient* —The blood under arterial pressure decreases in tension as it goes from the heart to the end artery A systolic pressure of 120 mm Hg in the aorta will be 90 in the terminal arteries a drop of approximately 20 mm Hg In the arteriolar bed this pressure drops to 32 mm The resistance to the arterial blood flow increases further as one reaches the capillary loops If it is possible for this blood to enter a venous channel where the pressure is lower than that of the arteriolar or capillary beds the tendency is for the blood to take this course of least resistance It is not unusual therefore especially in an extremity

for one to see a gangrenous digit develop In amputating such a part, the blood supply just proximal to it may make bleeding a serious problem This paradoxical situation often is difficult for the surgeon to understand but is fully explainable if one considers the falling pressure gradient, the increasing resistance to the blood flow and the readily available shunt to a low resistance fluid in the vein

*Skin changes* frequently are present There may be minor reddish discolorations, and some so-called port wine type of markings are indications of fistulas. Hemangiomatous lesions or masses of dilated vessels are suspicious. In any vascular skin change, a search for a congenital arteriovenous anastomosis should be made These congenital changes may be associated with other developmental anomalies It is characteristic of congenital arteriovenous aneurysm that many new connections open as others are closed

In large or multiple congenital arteriovenous fistulas, cardiac enlargement occurs as well, and if uncorrected, will progress to myocardial failure

*Venous Stasis Signs* —All the symptoms accompanying a reversed flow of blood in veins may occur, with pigmentation, edema, ulceration, dermatitis, and subcutaneous or actual hemorrhage In some of the smaller congenital fistulas, particularly those on the face, the appearance is the most serious problem In the larger congenital fistulas, the patient may complain of a buzzing sound, like a bee hive, as the blood whirls in the receiving veins The enlarged part, with the soft, warm skin containing the dilated vessels, is typical

In many patients, the fistula invades and affects nerves by pressure Other symptoms due to pressure depend upon the site involved.

**Altered Physiology of Arteriovenous Fistulas.**—There are certain pathologic changes which follow an arteriovenous anastomosis Holman[27–31] emphasized these points He described the arteriovenous fistula as a parasitic circulation, the short circuiting being the parasite which draws from the normal circulation

The physiologic changes are due to the short circuiting of the blood back to the heart caused by the fistula

**Explanation of Altered Physiology.**—The body attempts to compensate rapidly for this loss This compensation is similar to that response of the body to hemorrhage Fluids are drawn from the body tissues and the liver and spleen are drained of cells to restore at once the blood volume The suddenly lowered blood pressure slowly returns to normal. The diastolic pressure remains low, as the peripheral resistance permanently is lowered by the fistula The pulse rate may or may not decrease with compensation, depending upon the size of the fistula The total blood volume is permanently increased There is an increase in the quantity of blood entering the area of the fistula

The heart becomes distended and dilated The size of the heart varies directly with the quantity of circulating media The loss of a certain quantity of blood will markedly decrease the size of the heart The heart decreases in size in shock where a similar physiologic change occurs, as the blood is concentrated and therefore there is much less fluid in the circulation If the fistula persists, the quantity of blood entering it gradually

increases. This is a normal reaction inasmuch as the resistance at th fistula site is always much less than that at the peripheral capillary bed As this blood loss into the fistulous tract is continuous the blood volum in the normal circulation must be maintained by an increase in the tot volume There is therefore, an increased amount of work for the heart t perform Blood must be pumped through two separate and antagonisti systems

The degree of cardiac hypertrophy will vary directly with the size of th fistula When a fistula occurs in an extremity with fibrous tissue present point may be reached at which there will be no further distention of th tract and the peripheral resistance in both the fistulous tract and in th normal circulation will be equal

If the fistula is in the abdomen the pelvis or some area where there i little resistance the enlargement of the parasitic circulation may continu indefinitely There will be a gradual hypertrophy of the heart muscle an if this is slow enough cardiac failure may be postponed for many year

If the fistula persists for a time the heart muscle must hypertrophy I the fistula continues to enlarge *cardiac failure* eventually will result whe the heart no longer can force the blood peripherally with the continue loss through the abnormal opening

*Collateral circulation develops* around the fistula invariably if the fistula i maintained This collateral circulation permits successful surgical excisio of the fistula The blood sample from the vein of the involved area bein of arterial origin will show *a higher oxygen saturation content* than the sampl from another uninvolved vein

These cardiovascular changes described occur in any arteriovenou connection whether it is of acquired or congenital type, provided the defe is large The degree depends on the size, number and sites of the fistula

*Physiologic Changes in Arteriovenous Fistulas* —

1 Immediate Changes
   (*a*) The systolic and diastolic arterial blood pressure falls
   (*b*) The pulse rate increases.
   (*c*) The venous pressure proximally as well as distally increas depending upon the size of the fistula
   (*d*) There is an increase in cardiac output depending upon the si and location of the fistula
   (*e*) A temporary decrease in the size of the heart and arteri proximal to the fistula occurs due to the diversion of blood fro the arterial to the larger venous system
2 Remote Changes
   (*a*) A part of the circulating blood is permanently diverted from th normal capillary bed
   (*b*) The blood volume gradually increases dependent upon th amount of blood diverted
   (*c*) The heart and the artery and vein proximal to the fistula dila due to the distending effect of an increased volume of bloo which is attracted to the fistulous circuit because of its lessene resistance

(d) An extensive collateral circulation always develops around the fistula

(e) Hypertrophy of the heart follows due to dilatation, distention and increased work

(f) There is a recovery gradually from the lowered blood pressure, the pulse pressure being greatly increased [29,49]

**Pathology.**—The pathology depends on the size and site of the fistula The pressure of the arterial blood drives the venous blood back during systole, while in diastole, venous blood may enter the artery This arterial blood causes venous stasis At first, the arterial blood returns directly through the venous channels without being carried through the capillaries, and starvation of the periphery may occur This is gradually reduced by compensation The endothelium soon lines the connection and coagulated blood in the sac is not unusual The cells lining the opening of the fistula may be immature There may be fibrous tissue surrounding the opening, depending on the length of time it has been present When both walls of the artery and vein have been involved or the artery wall has been incompletely or completely transected, a pseudo-sac develops In these sacs the clot laminates and is surrounded by fibrous tissue which further strengthens it The contiguous structures also react to the fistula and adhere and strengthen or even form part of the wall

In some cases of congenital arteriovenous fistula, it is difficult to determine the site of pathologic transition between artery and vein In the resection, a large group of vessels in all histologic states, ranging from normal arteries and veins to immature structures with new growth-like cells, may be removed

**Prognosis**—The prognosis necessarily depends upon the site, size and type of the aneurysm and its treatment Rarely a fistula closes by itself Shumacker[64] found spontaneous self-cure in 8 of 245 (3 3 per cent) In only 2 per cent was the end result satisfactory Only one of Callander's 497 patients had spontaneous resolution [5] Winslow[67] had no non-operative closures in 19 patients Of the author's 136 arteriovenous fistula operations, none has been spontaneously cured where the fistula was of sufficient size to have produced a bruit In several instances, bruits have disappeared but have later recurred Reports of spontaneous cure, therefore, may be remittances In small fistulas diagnosed only by the finding of arterial blood in dilated veins, cures occur more readily and have been observed even after incomplete excision This failure of fistulas spontaneously to close probably is due to the low pressure on the venous side and the reduced arterial lumen distal to the fistula as well as the arteriolar and capillary bed resistance.[33] This is in contrast to arterial aneurysms where spontaneous abatement is less unusual An occasional complication is the *development* of subacute bacterial endocarditis which makes the prognosis for recovery poor [38,42,60,61] The size and duration of the fistula regulates the degree of cardiac hypertrophy and thus directly affects the prognosis Where cardiac hypertrophy is marked, complete recovery of the heart cannot be expected A sudden operative closure of the fistula may throw the entire load of the increased blood volume on the weakened heart and cause cardiac failure. If the fistula has been open a long time, venesection at the time of

operation will prevent this complication The small arteriovenous fistulas described as arterial varices and having the symptoms of varicose veins have a good prognosis as far as life is concerned They tend to recur locally after operation and thus prognosis for cure is not good Some of the borderline arteriovenous cases such as congenital hemangiomata, are difficult to alleviate The invasiveness of many of the congenital types makes their excision at times impossible Where they invade important parts of the body they may kill by pressure or by ultimate hemorrhage Thus we have seen arteriovenous fistulas invade the spine and cause paralysis At least two fatal hemorrhages into the thoracic cavity were traced to ruptured arteriovenous fistulas.

The prognosis for fistulas in the brain for example is serious. Pulmonary arteriovenous shunts are extremely serious and are the cause of some of the blue babies Pneumonectomy has been required for cure [3, 11, 22, 72]

**Treatment.**—EARLY THERAPY—The early treatment of acquired or congenital arteriovenous fistula consists of stimulation of the collateral circulation Time plays an important part in the development of collateral circulation The mere existence of the fistula stimulates the collateral circulation Local ischemia distal to the fistula however may force an earlier operation

*Tests for Adequate Collateral Blood Supply*—A practical point is to observe the blood supply to the part distal to the fistula with the fistula occluded The Matas[44] reactive hyperemic test is of value to determine if the collateral circulation is adequate The original test has been modified by many surgeons A simple method is as follows

Elevate the limb and remove the blood from the part by bandaging Apply a tourniquet above the lesion remove the bandage, and with digital pressure completely close the fistula site The fact that the fistula is correctly closed by the digital pressure should be checked by a stethescope Release the tourniquet If the collateral circulation is adequate the part distal to the fistula will progressively improve in color and temperature If such change does not occur in two minutes [44] the possibility of an inadequate collateral circulation exists Since the operation to excise the fistula will further interfere with collateral circulation this test fails sometimes to correctly prognosticate the adequacy of the arterial supply

In general a delay of three to six months is indicated to permit collateral circulation to develop At the operation time small vessels which are encountered in the dissection should be preserved if possible until the lesion is exposed One should observe the effect of a temporary restraining tape on all of the vessels entering the fistula before dividing them The distal part should be so draped that it can be inspected at any time. In these patients interruption of the sympathetic nervous system to the involved part is important during the preliminary stage

1 **Treatment of Acquired Type**—Acquired arteriovenous fistulas are usually large and require surgical intervention

From four to six months time should be allowed after the fistula develops before an operation is performed provided (1) that there is no emergency from the hemorrhage or pressure standpoint (2) that there is no evidence of rapidly occurring cardiac failure and (3) that there is no evi

dence that too much blood is returning through the vein and thus interfering with the nutrition of the part distal to it Gangrene has followed such diversion of circulation Such an event dictates an earlier operation During this four to six months, the collateral circulation will develop. In most patients a preliminary surgical sympathectomy improves the arterial supply and insures a better result

The operation consists of the exposure of the vessels proximal to, distal to, and at the site of the anastomosis

**Preoperative Preparation.**—In those few patients where immediate operation is required, the injured area should be carefully shaved and washed with one of the sulfonated detergent soaps (with Hexachlorophene) Antibiotic therapy, gas gangrene and tetanus antiserums should be administered

In these patients, the surgeon must be prepared to overcome severe hemorrhages or to combat sudden shock Where there has been considerable blood loss externally or internally, a small additional hemorrhage may be sufficient to throw the patient into a profound shock With hemorrhage, the threshold for shock is lowered

**Anesthesia** —*Spinal Anesthesia* —This is the ideal anesthesia for the lower extremities This may be given with procaine for relatively short procedures, pontocaine or a mixture of the two for longer ones, and continuous spinal with procaine in extended procedures

For all operations in the thorax, near the diaphragm or in the neck where danger of injuring the pleura exists, *intratracheal* anesthesia should be used The positive pressure, thus available, decreases greatly the dangers and has been one of the important advances of modern cardiovascular surgery An unnoticed injury to the pleura may result in a tension pneumothorax The house staff should be alerted to this possibility and the necessary implements to evacuate the air in such an event must be available at the bedside after the operation

For the upper extremities, a *local* or *general* anesthetic can be used For the head and neck, local anesthesia is advocated unless the dissection may involve the pleura This infiltration or block anesthesia permits the cooperation of the patient in determining any cerebral symptoms In questionable cases, intratracheal anesthesia is suggested

*Hypotension Anesthesia* —It has been found that in many of the arteriovenous fistulas and aneurysms the blood loss from trying to dissect, divide and tie the involved vessels has interferred with success At times the operation must be terminated due to the patient's status and often the operation is incomplete due to this fact and the technical difficulty of operating under these conditions The anesthesia increases the problem when there is straining or deep breathing In these patients the initiation of a hypotensive state in which the blood pressure is dropped to a level where bleeding is reduced has improved the mortality rate and the results In addition, some aneurysms previously considered incurable have been successfully eradicated There are many such operations reported, particularly in the brain The blood pressure may be lowered in two ways

(*a*) *Total Spinal Anesthesia*[68]—This method requires sufficient anesthesia to cause a drop in the blood pressure to below shock levels, i e, 20

to 40 mm Hg At this level the blood circulates under low pressure but the bleeding coincident to operating on the vascular tumor is not present Morton[45] and Jonnesco[37] have reported on this technic the former as early as 1900 The effect on the medullary centers and motor and sensory cranial nerves depends on the concentration and volume of novocain used A report by Griffith and Gillies[34] showed no permanent central nervous system lesion in 84 instances. This method will be further improved (See Chapter 3 on Anesthesia)

(*b*) *Hypotensive Drugs* —The author has observed the beneficial effects of hypotensive anesthesia by drugs as used by the British and French surgeons The same methods in arteriovenous fistulas and other vascular operations have been used innumerable times in the last few years With care and judgment operations on vascular tumors previously not eradicable are now possible The hypotension is produced by drugs (25 mg of hexamethonium chloride) This is a variable dose depending on the size weight and physical status of the patient

**Immediate Post Injury Surgery** —The surgeon should operate at once if necessary to control the hemorrhage or to relieve pressure from the hematoma An immediate operation is justified also when there is evidence of circulatory loss to the extremity or nerve pressure

The incision should be made proximal to the site of entrance of the missile or wound causing the fistula Control of the vessels proximal and if possible distal to the wound should be gained before disturbing the clot Such control may require even opening the chest to pass a tape around the subclavian or innominate arteries for upper extremity injuries It may be necessary to divide the inguinal ligament and dissect retroperitoneally to tape the iliac vessels for femoral lesions The infra foramen approach to the vertebral vessels was an addition developed during World War II [41,44] The author advises the routine use in the operating room of an excellent anatomy book with good pictures No matter how good an anatomist the surgeon may believe himself to be there will be times when identification of a structure is difficult and time-consuming In the operating rooms at the U S Naval Hospital at St Albans during World War II white stands similar to those which hold bibles in churches were standard equipment The anatomy book was always opened at the right page This is important particularly where traumatic wounds have altered the normal anatomical picture. Technical errors will be minimized in this way The site of the injury then can be explored and all clots and dead tissue débrided and involved vessels exposed

In operating the same general surgical principles are to be followed as observed in any operation on a newly formed traumatic wound Obvious dead tissue should be removed formation of pockets prevented and all nerves carefully avoided or if injured identified and repaired

If it is at all feasible the artery is restored by suturing it with the atraumatic silk suture technic from the vein side as described on page 417

The vein then is opened further and inspected for thrombosis ligated and resected Should a thrombus be present in the vein, it should be removed with forceps or with aspiration until such time as a free flow from the proximal end of the vein is obtained The vein then should be divided

and ligated with a transfixing suture sufficiently proximal to the main wound so that the secondary necrosis and slough from the wound will not include the vein or cause a continued thrombosis

Efforts to repair the vein are unnecessary and dangerous due to possible pulmonary embolism

In the presence of *gross infection,* arterial sutures should be attempted only if it is apparent that the part will be lost if the artery is ligated If the vessels are ligated it should be done in a viable area and at a safe distance from the fistula

Antibiotic therapy locally and systemically is indicated

General surgical skill is required inasmuch as both fractures and injury to nerves or tendons may occur concomitantly in acquired arteriovenous fistulas

**Technical Points Applicable to All Arteriovenous Operations.**—The management of arteriovenous fistulas requires general surgical background and knowledge The finest aneurysmorrhaphy will fail if the wound is infected or thrombosis occurs in the vessel Certain points are important

*Infection* is most important as a vessel operated upon in its presence may slough with hemorrhage The sacrifice of dead tissue is fundamental and must be performed *Blood loss* must be replaced, nearly liter for liter The *incision* in general should be in the line of the vessel and should begin above and end below the site of the tumor If the incision crosses a joint it may be made in the form of a "Z" or "S" A *sterile stethescope* must be available to test whether all bruits have been corrected Sufficient hemostats and other *equipment* for possible need must be procured The operation requires *skilled assistants* and the trained team increases the operative efficiency *The time of operation is important* The optimal time of four or more months should be permitted unless there are signs of cardiac failure, peripheral ischemia, an increase in the size of the fistulous mass or hemorrhage

To these must be added the selection of the correct operative procedure The operation for an arteriovenous anastomosis is not a dressing room or front line procedure The following operations may be indicated

In the *traumatic type of aneurysm,* many can be resected with end to end anastomosis, others can be excised with repair of the defect in the artery with sacrifice of the vein, a few can be bridged by an analogous vein or homologous artery graft, and the rest must be obliterated as in the congenital ones In the aneurysms due to *congenital defects* in closure, nearly all are of a multiple occurrence and these respond best to wide resections In the occasional single congenital defect some restorative or reparative procedure may be considered In those due to *degenerative diseases* the treatment depends on the individual circumstances Sometimes it is possible to restore such an aneurysm by repair or anastomosis but the poor type of tissue does not lend itself well to suture The use of analogous grafts with veins or homologous arterial grafts frequently is not feasible In the main, excision of the mass with multiple ligations is effective In some, a muscle implant can be inserted into the well-defined sac Chemical obliteration of small aneurysms of this type has been feasible in our Clinic in only 3 per cent

**Operative Procedures for Acquired Arteriovenous Fistulas.**—Where the operation is not an emergency procedure an arteriogram with the dye injected proximal to the fistula is of the greatest aid. The site of the fistula or fistulas will be shown, the collateral circulation can be demonstrated and often the decision on the type of operation will be readily made with such roentgen delineation. Of our 120 arteriovenous aneurysms 24 per cent of them were due to degenerating diseases.

(1) *End to End Anastomosis*[43,44].—The ideal treatment is to close the opening in the artery, tie the vein and remove the sac. At times, particularly in the region of the joints, the connection may be divided and the

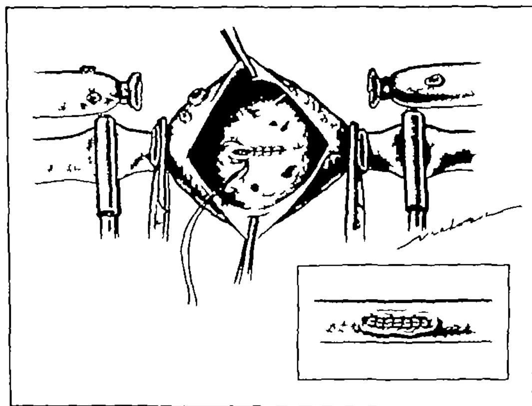


Fig. 120.—The repair of an arteriovenous aneurysm through the sac. The vein has been resected, *the sac opened and the opening from the artery into the sac is being* sutured. Insert shows sac used for reinforcement, excess cut away. This type of repair is applicable in many more patients today than in previous years.

artery anastomosed by end to end suture. Rerouting of the vessels and flexion of the joint often will provide the required length of segment. After such anastomosis this vessel can be lengthened gradually by extension.

(2) *Repair of Artery Through Sac or Vein Wall.*—The closure of the fistula by suturing through an opening in the vein, as advocated by Matas[44], has been effective in many instances. The vein then may be resected. We have performed this operation 22 times with success.

(3) *Excision of Fistula with Analogous Vein or Homologous Artery Graft.*—The excision of the aneurysm or fistula and the insertion of a

vessel to bridge the defect theoretically answers the problem Unfortunately it is not practical all of the time In only 2 per cent were we able to graft the defect It is apparent that only an analogous vein graft from the patient is possible because of the obvious dangers to the donor site with the removal of a segment of artery Homologous grafts are practicable For technic see pages 340 to 342

(4) *Multiple Ligation*[55,56a]—This treatment of the affected artery and vein, with resection of all the involved vessels and sac, often is the most satisfactory surgical treatment This eliminates the source of trouble, and since the collateral circulation usually is sufficient, it is a most effective treatment



FIG 127 —Patient from Figure 128 immediately postoperatively Parasitic circulation had resulted in atrophy of the distal leg Sympathectomy used as adjunct treatment

(5) *Ligation and Excision of Involved Vessels* —The usual term, quadruple ligation for arteriovenous aneurysm, is a misnomer based on the supposition that there are only four involved vessels, the proximal and distal artery and vein If such a fistula has been present any length of time however, it is found that there are many more than four vessels involved There may be a main artery and vein entering and leaving the vascular mass, but there are also innumerable collateral vessels involved. These develop at least within two months' time

This large number of vessels at times is confusing to the surgeon who expects to expose four vessels resect them and have complete control of the area The innumerable vessels present with branches opening into the mass account for the unexpected and severe hemorrhage that occurs

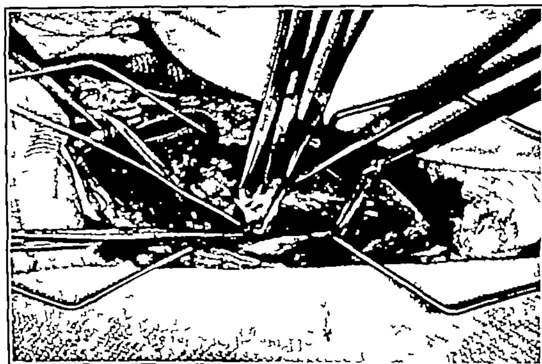


FIG. 128.—Operative repair of traumatic arteriovenous aneurysm. Repair of artery through sac. Small arterial opening sutured with fine silk. Sac used to reinforce suture line.

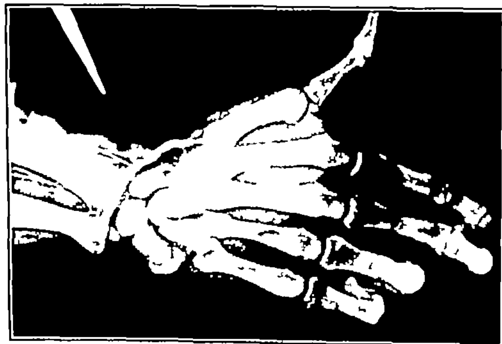
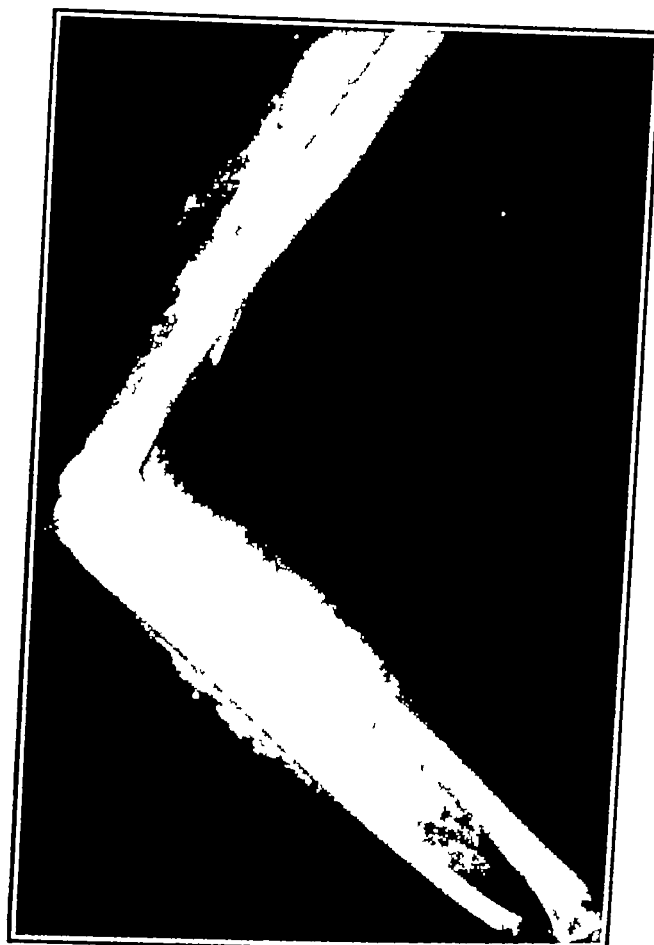


FIG 129 —Radial arteriogram demonstrates innumerable arteriovenous connections. Trauma precipitated the swelling, pain and dilated vessels. Treatment excision of all venous components.

Fig. 130



Fig. 131



Figs. 130 and 131. Congenital arteriovenous fistula involving all the vessels of the arm with hypertrophy. Arteriogram shows blood returning through parasitic circulation. Repeated resections only temporarily effective. Ligation and resection of subclavian artery reduced all pressure and effected cure. Note tremendous number of arteriovenous connections.

While control of a major vessel proximal and distal to the mass will help to a certain extent in eliminating excessive hemorrhage the surgeon must expect to clamp and divide many more vessels as the lesion is exposed This is true always in the congenital type and also in the acquired type, depending on the length of time it has been present

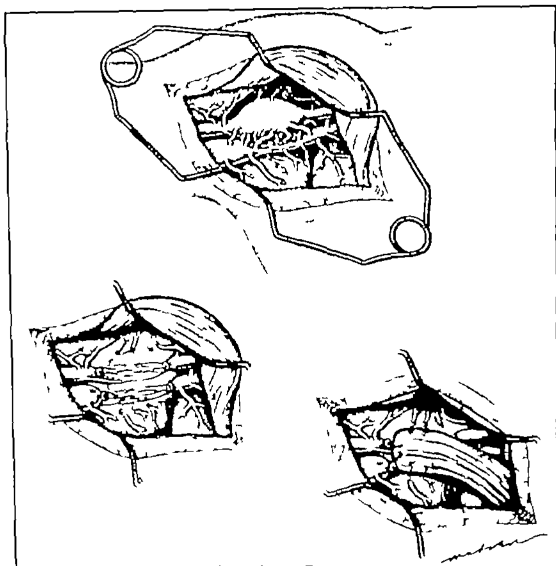


FIG 132.—Arteriovenous aneurysm in the axilla. Multiple connections between the artery and vein. Treatment excision of the area involved with a muscle implant in the defect All vessels entering the sac area were ligated and excised (Pratt courtesy of Am. J Surg.)

At times as many as two and three hundred vessels must be divided in exposing and eradicating an arteriovenous fistula. The surgeon must be prepared for this contingency both with instruments and skilled assistants.

The mass is mobilized at one end The division of the main vessel will help in the dissection The vascular tumor then is elevated and each vessel as it enters the area is divided. The mass and all its entering vessels

are resected Each vessel is separately ligated with fine silk sutures The main vessels should be transfixed with their ligation

(6) *Obliteration of Sac with Muscle Implant* —In some fistulas, the collateral circulation is inadequate for excision of the sac In these instances, as in arterial aneurysms, the necessary collateral vessels for the circulation distal to the fistula have developed in the sac and the preservation of the sac is required In such cases, the vessels entering the sac are ligated and each opening into the sac sutured. The sac is then obliterated by a muscle implant and imbricating sutures as in arterial aneurysms (See p 377.)

(7) *Other Procedures* —It is impossible to detail all of the operative technics used for acquired arteriovenous fistulas as each case varies in its surgical requirements In this respect, the surgeon often must develop ingenious methods to handle the vascular problem as it arises, using these fundamental principles as a basis.

(*a*) Wherever possible, restoration of the artery is the treatment of choice

(*b*) If the fistula has been present at least three months, sufficient collateral circulation will be present in most cases to permit excision This should be checked by temporary occlusion in the operative field and observation of its effect

(*c*) In such event, every involved vessel must be excised.

(*d*) If the fistula is of recent origin, some method of re-establishing the arterial circulation may be necessary for the survival of the part Of the available methods, end to end anastomosis or repair are the best procedures, with vein grafts or arterial bank grafts alternative possibilities.

(*e*) In some fistulas where there is a well-developed sac, simple excision of the vessels involved is not sufficient In such cases, obliteration of the sac after ligation of each vessel will be adequate and will help to maintain the collateral circulation

(*f*) Delineation of the fistula and collateral circulation by arteriography is a useful and often necessary prerequisite to successful surgery

(*g*) Sympathectomy often helps the peripheral circulation

**Use of Tourniquet.**—In the simple traumatic type of arteriovenous fistula or aneurysm, the use of a tourniquet will save time and blood loss Its use must be reserved for those in the younger age group, as application of a tourniquet in the older patient may injure the artery, especially if any sclerosis is present In the congenital or degenerative disease type of aneurysm, unless there is an unusual reason which makes the use of a tourniquet imperative, we prefer to perform the operation without a tourniquet The use of a tourniquet may be time-saving and may prevent considerable hemorrhage, but it has certain disadvantages A tourniquet palsy may follow Again, certain arteriovenous connections are not visible or identifiable when the operation is performed with a tourniquet At times, these vessels are so small that they collapse completely when the tourniquet is applied and their identification and resection is difficult

In many cases there are so many of these congenital connections so near the skin that although they can be controlled temporarily with Ellis clamps, when the clamps are removed the hemorrhage from these areas in and directly under the skin is enormous As a result, each connection has

to be separately identified, dissected free, and ligated—a most difficult procedure because of the fact that they tear easily

In certain cases where the number of collateral vessels is enormous it is wise to apply a tourniquet, releasing this prior to the time that the closure is made to permit the identification and resection of other vessels which have not been observed

At other times the application of a tourniquet causes so much venous congestion that the operation is more vascular than without any constriction

*In certain instances, such as in the acquired type of arteriovenous aneurysm* following a gunshot wound where there is usually damage to other structures it has been impossible to get adequate tourniquet application



FIG 133 —An extensive arteriovenous aneurysm involving the ulnar artery (arteriogram) Most of the vessels of this arm were involved. Note ischemia of some of the fingers. (Photograph courtesy of Dr Irving S Wright.)

If the tourniquet is used it should be of the pneumatic type applied so as to make the field entirely bloodless.

2 **Treatment of Congenital Arteriovenous Fistulas** —The treatment of congenital arteriovenous aneurysm is difficult because the fistulas are multiple In most cases the patient is operated without a tourniquet Fistulas may be left if the blood supply is shut off during the procedure.

A *sterile stethoscope* should be at hand and should be used frequently to locate the site of the bruits and check on their obliteration.

Heroic excisions of the involved vessels often are necessary for success The vessels may have so invaded the surrounding tissue that their removal from this tissue alone is impossible. Such vessels invade muscles, fat, nerve tissue or glands. It may be necessary to excise the surrounding tissue en bloc in order to eliminate the fistulas. When the involved tissues have

been excised, a few leading vessels below or above the vascular mass will be found, which are the feeding ones These are of arterial origin

The danger of local necrosis and gangrene after such excisions must be faced and coped with in each case on the basis of the surgeon's experience

In one case it was necessary to remove the entire radial artery distal to the elbow and one-half of the ulnar artery The collateral supply was sufficient and there was no gangrene Carrel[6] devised a clamp to be applied to the artery to determine if its ligation would interfere with circulation. Spasm may interfere with such tests A soft tape or rubber band serves the same purpose with less trauma Where the question of the adequacy of the circulation is imperative, temporary obstruction must be applied for a longer time For example, resection of the internal carotid artery may result in hemiplegia and death in from 10 to 30 per cent of the cases[41, 51]

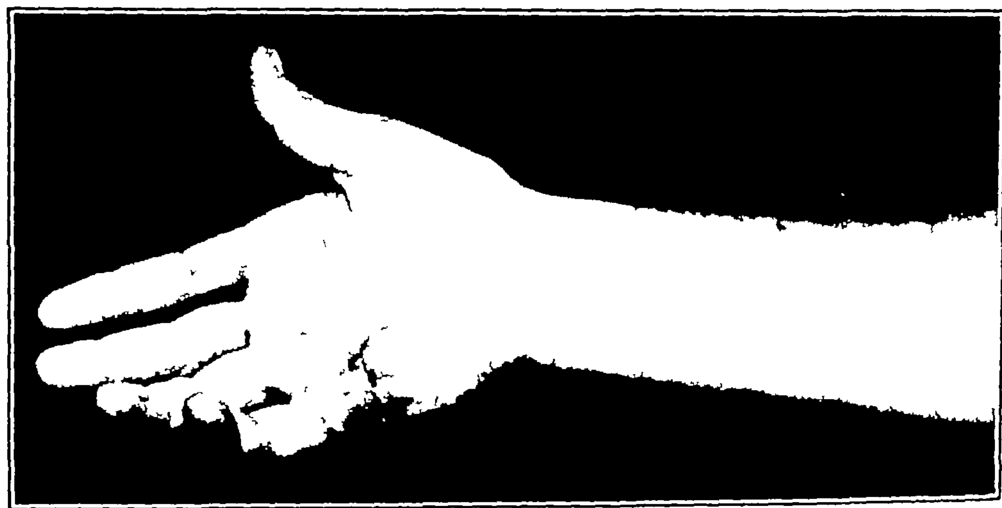


FIG 134 —Large arteriovenous aneurysm of the hand of congenital origin Changes in the skin had been noticed since birth During World War II the patient dropped an ammunition case on his hand, precipitating an aneurysm necessitans An egg-shaped pulsating mass was protruding through the hand Treatment consisted of excision of mass

This incidence is less in patients with fistulas due to collateral circulation development, but the possibility still exists Dandy[10] believed that a ten-minute obliteration without cerebral changes indicated safety Others have not found this to be true It is suggested in such cases that the vessel be obliterated for from forty-five minutes to an hour with careful observation of the reactions of the patient and his physical signs If the fistula has existed a long time and it is important for it to be eradicated, it can be excised thereafter with relative impunity In any questionable case however, it is safer to tie the artery in continuity and be prepared to remove the ligature should cerebral signs develop If removal of the ligature is not sufficient to correct the cerebral signs, the artery should be opened and any thrombus extracted

Suction can be applied up the artery into the brain

**Subsequent Fistulas** —In the congenital type of arteriovenous fistulas there may be other connections not patent at the time which may open spontaneously after the closure of the fistulas present They may open due to the rise in local pressure which follows the closure of the other fistulas In these cases the openings between the arteries and veins have been temporarily occluded or congealed but there is an inherent weakness in the walls *The lining of this closure may be only a single layer of endothelial cells* When the pressure adjacent to it is increased by the closing of other fistulas this long-delayed opening occurs.

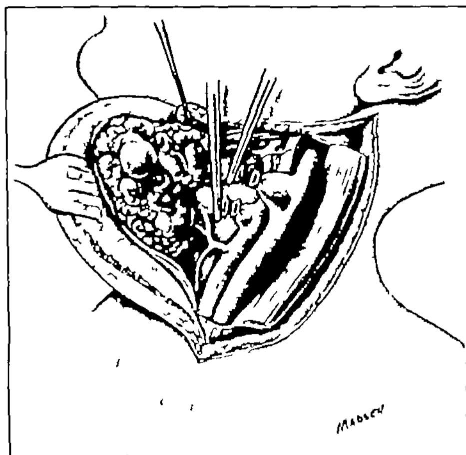


FIG 135 —Extensive arteriovenous aneurysm of the neck. This aneurysm arose from the external carotid artery on the left side and then involved most of its branches and the entire venous tree in the neck. Treatment was surgical excision (Pratt courtesy of Surg Gynec & Obst )

The possibility of the appearance of new openings should be explained to the patient before operation in congenital arteriovenous connections so that a new opening will not be considered a recurrence due to faulty operative technic

In one instance approximately 14 congenital connections were excised in one arm A year later the patient returned with 5 new ones After subsequent removal of these the patient has remained well

Some of the residual veins may be sclerosed subsequently if one is certain there is no arterial connection to an essential part of the body

**Operative Procedures for Congenital Arteriovenous Fistulas.**—The operative technic must vary somewhat with the site of the fistulas and the local conditions found, *i e*, the number of vessels involved and the area invaded by these vessels The operation of choice is a resection of the fistula and the veins involved with restoration of the artery This is not always possible, however, and in most cases, multiple ligations, with excision of the fistula and mass of arteries and veins with or without the adjacent tissues, usually are employed

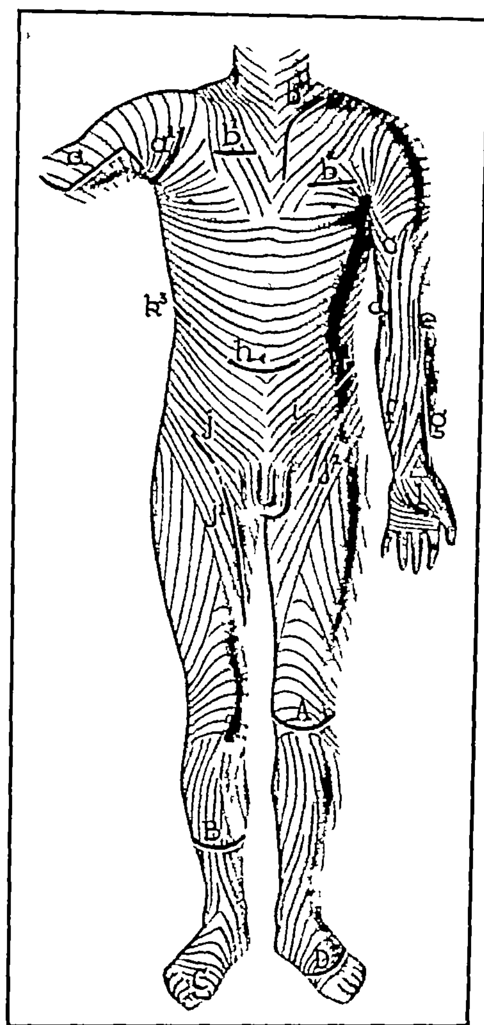



FIG 136

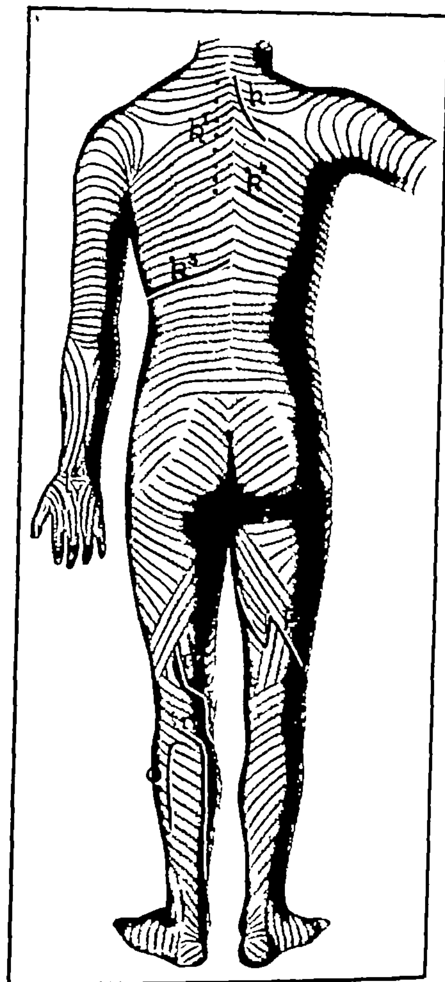


FIG 137

FIG 136 —Skin incisions Modification of Henley's lines showing the line of skin cleavage and the incisions least likely to cause contracture or retraction at the joints

FIG 137 —Showing the posterior incisions in the lines of skin cleavage

Adequate control of all vessels proximal and distal to the fistula is important before excision of the sac The collateral circulation is so adequate that proximal control alone will not prevent an alarming hemorrhage from the collateral vessels or from the distal end

In the treatment of congenital arteriovenous connections, it is not uncommon to find more than one artery involved In some of these fistulas, it is wise to expose the artery proximal to all bruits and then

excise the veins accompanying it distally. Any branch coming off the artery which is tortuous or thin walled should be excised. All vessels entering the vein are resected. Many times all the veins encountered in such a procedure must be resected.

Since blood vessels accompany nerves, these latter must be identified and protected against injury.

*Incision.*—If it is necessary to have an incision at a joint, it should be of a Z or S type if possible, to decrease the amount of constriction and

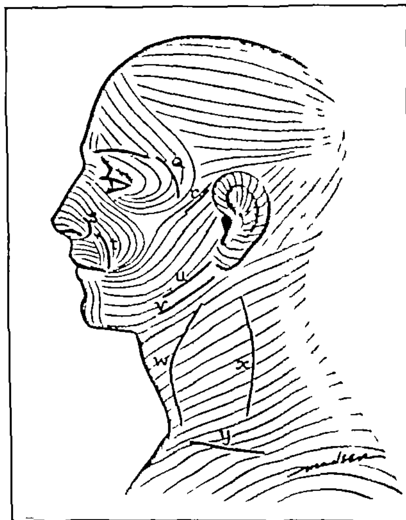



Fig. 138.—Skin incisions and lines of cleavage for the head and neck.

scar or scar retraction occurring at the joint. Types of incisions are shown in Figures 136, 137 and 138.

*Bone Resection.*—It is frequently necessary to resect bone to reach a fistula.[17] One should not hesitate to remove subperiosteally all or part of the clavicle, sternum or ribs to reach the subclavian or innominate arteries.[18,19,20,21,22] The entire scapula has at times been sacrificed.

(1) *End to End Anastomosis.*—In some patients, especially those in whom it is necessary to operate early when collateral circulation is in

adequate, end to end anastomoses may be performed Unfortunately, these patients are few in number.

(2) *Repair of Artery Through Vein Wall* —This is theoretically possible, but so rarely is a single congenital fistula seen that its practical application is limited

(3) *Vein or Artery Graft* —The use of grafts of an analogous vein or homologous artery is possible but practically rare These congenital anastomoses are so infantile in cell structure that they are rarely single or localized There will be the occasional one which will respond to such therapy, but this will be the exception and not the rule For technic see pages 340 to 342



FIG 139 —Shows an arteriovenous aneurysm at the base of the tongue This was expansile and pulsating but because of its location it was difficult to excise It was treated satisfactorily by local injection (Courtesy and permission of Dr L K Ferguson and Surgeon General, U S Navy )

(4) *Multiple Ligation of the Involved Vessels* —This therapy is effective in many of these patients If applicable, all the involved vessels should be ligated and, as far as possible, excised Small collateral vessels then carry the circulation load Unfortunately, these too may have or may develop fistulas

(5) *Ligation and Excision of the Vascular Mass* —This may be possible, especially where the vessels involved are not essential primarily for the life of the part

(6) *Endoaneurysmorrhaphy with Muscle Implant* —In many patients, the operative technic of endoaneurysmorrhaphy of Matas used in arterial aneurysm has been necessary and effective in saving the extremity The sac may contain so many necessary collateral vessels that it must be preserved (See Fig 132 )

An operation in which each vessel entering the area is ligated and a large contiguous muscle is inlaid in the area where the fistula existed has been reported by the author[34] as curative in some cases (See Fig 109 page 377 )

Obliteration of these sacs by suture alone is technically difficult and in some instances impossible In these cases our modification consists of inlaying a muscle transplant to provide a core for the sac. We have used this method in 7 per cent of our operations for arteriovenous aneurysms

*Importance of Differential Diagnosis Between Arterial and Arteriovenous Aneurysm* —The importance of differentiating an arteriovenous fistula

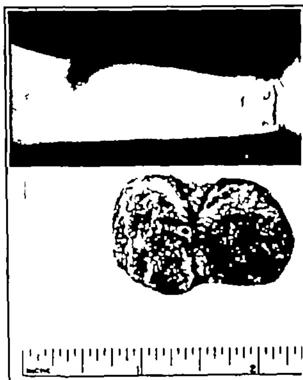


Fig 140 —Aneurysm at the bifurcation of the brachial artery into the radical and ulnar arteries. This had been present for one year and trophic and Raynaud-like symptoms were occurring in the hand.

*Lower* Pathologic section of tumor removed in upper illustration

from a simple arterial aneurysm should be understood If a fistula remains after the resection of the artery gangrene often results This occurs because the collateral circulation on which the life of the part depends instead of going past the ligated point to nourish the extremity goes at once through this fistula and returns to the heart

(7) *Chemical Obliteration* —In some of the smaller fistulas appearing particularly on the face, where the collateral circulation is excellent the use of a sclerosing solution may be sufficient to control the condition and in some cases is curative This applies to the type of arterial varix seen around the eyes and mouth The use of some tourniquet like device such as a rubber ringed baby pacifier to hold the sclerosing solution at the desired site and produce more effective sclerosis is unnecessary This practice may be dangerous as the pressure may produce a slough or an

embolism may follow its release. This type of sclerosing treatment must be used only in properly selected individuals. It is emphasized that sloughs are not uncommon.

(8) *Proximal Artery Ligation*.—In an occasional congenital arteriovenous aneurysm the arterial pressure in the arterial component is so great that new areas of anastomosis break open after the ones present are closed. Since it has just been stated that ligation of an artery when a fistula exists will cause gangrene, it is incongruous to state the opposite to be true. In some patients, however, the pressure in the parent artery may be so great as to continue to "blow open" new fistulas as fast as the old ones are closed. Some surgeons have submitted such patients to amputation. Before doing this irreversible procedure we ligate the artery proximal to the fistula to cut down the degree of pressure. In many such patients we have had the happy result of having the bruits disappear. While gangrene is a possibility in such ligations, the step is justified before a major amputation is performed. Where gangrene follows, there may be more of a stump left than would have been saved if the amputation was performed electively without the ligation. The artery may be ligated in continuity and later divided.

In the small arteriovenous fistulas described by the author as "Arterial Varices" (see pages 589 to 595), chemical obliteration has been effective after the main connections have been surgically divided. Such patients should be kept under observation and injected with small amounts of the sclerosing solution whenever dilations indicate that a small opening has occurred.

3. **Treatment of Arteriovenous Fistulas and Aneurysms of Degenerating Disease Origin.**—These connections are rare but occur due to the degeneration of the involved vessels due to such diseases as arteriosclerosis, lead poisoning, tuberculosis, syphilis, etc. The most common cause is arteriosclerosis. In many such lesions the artery first gives way, as in an arterial aneurysm, and the vein is secondarily involved when it becomes attached to the vascular tumor and is eroded. Resection of such an aneurysm is possible but rare. At times it can be resected and/or obliterated, as has been described under other treatment of fistulas.

**Certain Factors of Physiopathologic and Therapeutic Importance in the Treatment of Arteriovenous Fistula.**—1. An arteriovenous fistula will cause an enlargement both in the soft tissue and the bone of the part of the body involved.[35]

2. The reasons for the development of collateral circulation around an arteriovenous fistula are due to the ready access of blood to the fistula site by a retrograde circulation through a patent distal artery and its branches.[33] This negates the theories that some chemical substance in ischemic tissue initiates the collateral circulation or that the requirements of the tissues beyond the fistula development excite the collateral circulation.[58]

3. "End pressure" beyond the fistula is proportional to the resistance beyond that point. Thus it will be greatest in the ascending aorta where the entire peripheral resistance exists, and it will decrease from this point distally.[33]

4. The physiologic changes attendant to an arteriovenous fistula are due to the fact that the fistula creates an area of low peripheral resistance

Thus the blood at arterial pressure will enter a low pressure or venous circulation more readily than it can enter an arterial or capillary bed area[30,31,34,35,38,39,40,49,50]

5 Following the development of an arteriovenous fistula cardiac enlargement ensues While this enlargement may vary with the location size and age of the fistula the size of the vessels involved also is a factor Enough evidence is at hand to make it certain that these changes occur and are reversible by surgical therapy if the fistula has not existed too long

6 The cardiac output depends upon the size of the fistula The amount of excess blood pumped through the heart thus varies but is greatly increased in over half the patients After the fistula is closed, the output decreases.

7 In 50 to 85 per cent depending upon the size of the fistula the heart rate and heart size can be decreased if the fistula is closed[22]

In the majority of the patients the blood volume decreases after removal of the fistula relative to the size of the fistula

*Amputation for Arteriovenous Fistula* —In a few patients the arteriovenous fistula cannot be cured due to local conditions In others the cardiac status and failure forbid further attempts In these patients a major amputation may be necessary and lifesaving The number so treated should be minimal The major amputation is an admission of therapeutic failure That many patients lose a limb because of a defeatist attitude toward further surgical attempts is a certainty Before irreversible amputation is performed it is suggested that all therapeutic efforts be exhausted and that consultation by accepted specialists in this field be obtained

**Therapeutic Formation of Arteriovenous Fistulas and Their Use** —
1 *Lateral Arteriovenous Anastomosis to Increase the Collateral Circulation in Arterial Obliterative Disease* —This point has been discussed under arteriosclerosis and details can be found on pages 210 to 212 The value of such therapy is theoretical at this stage. While collateral circulation develops in every instance after a lateral anastomosis of an artery and a vein ischemic changes occur distal to such an anastomosis in a high percentage of cases If the fistula is of sufficient size, distal gangrene has occurred Its employment is restricted to those patients in whom gangrene is inevitable under a more conservative therapeutic regimen In the fistula created to increase the blood supply to a leg a three-way technic has been used The proximal vein is ligated and resected (See page 211 )

2 *Creation of an Arteriovenous Fistula to Reduce Pressure in the Artery Proximal to the Fistula* (*i e Aneurysm* ) —This operation is based on the hydrodynamic law that if the velocity is increased in a limited area in a circulating media within a tube the wall pressure is reduced Thus when an aneurysm develops in the wall of the artery the velocity of blood flow decreases and the wall pressure increases This is the basis of the Babcock operation for aneurysms This procedure originally was developed for aneurysms of the ascending aorta and has been of some value In this operation (see pages 385 to 386) the arterial blood under high pressure in the carotid artery distal to the aneurysm is shunted by end to end

anastomosis to the proximal end of the jugular vein, thus immediatel returning it by the superior vena cava to the heart The rate of flow i the carotid artery immediately distal to the aneurysm is increased I addition, the peripheral resistance is eliminated by circumventing the bloo flow immediately back to the heart The pressure is decreased in the aneu rysmal sac Babcock's experience with this operation demonstrated marked decrease in the mortality rate, relief of pain, and extended longevit in these patients The operation has a physiologic basis and its possibilitie in other fields have not been sufficiently explored [1]



Fig 141 —Carotid artery-jugular vein anastomosis to revascularize the brain Patient mentally retarded Anastomosis taken down after three years Apparent improvement in patient's mental status

3 *Revascularization of the Brain* [25,45,46,47] —The occurrence of convulsive disorders, spastic paralysis and mental retardation is not unusual in the developmental abnormalities seen in pediatric practice While some brain injuries follow natural birth, Yannet's[71] report on 1,330 abnormal children demonstrated only a 3 per cent brain injury as a cause, while 38 per cent showed familial defects in mental background and 22 per cent were of congenital source The possibility of birth injury including lack of oxygen as a major etiologic cause has been advanced by Cole [9] Modern efforts to make shorter and easier labors for expectant mothers probably increase the trauma to the child's brain One has only to look at the necessary configuration produced in the baby's head in primipara to realize that intracranial pressure on the child's brain must be greater if the force of labor is increased The early signs of spastic paralysis and delayed respiration may give secondary and permanent changes. Premature separation

TABLE 36.—SUMMARY OF ARTERIOVENOUS ANEURYSMS SURGICALLY TREATED
(126 OPERATIONS IN 87 PATIENTS)

| *Etiology* | *Site* | *Main Symptoms* | *Prognosis* | *Treatment in Order of Value* |
|---|---|---|---|---|
| 1 Trauma—24%<br>Gunshot<br>Stab<br>Auto or plane | Any vessel<br>Most often<br>1 Arms<br>2 Legs<br>3 Head and neck | Thrill and bruit<br>(Systolic and diastolic)<br>Tumors<br>Dilated veins<br>Color changes | Good with adequate operation | 1 Resection with anastomosis of artery<br>2 Excision with repair of artery<br>3 Obliteration |

TABLE 37

| *Etiology* | *Site* | *Main Symptoms* | *Prognosis* | *Treatment in Order of Value* |
|---|---|---|---|---|
| 2 Congenital—70%<br>Onset at birth or puberty | Any vessel most often arms, legs | Same but widespread Few no bruits | Guarded generalized disease tends to recur | Multiple resections may require amputation |

TABLE 38.

| *Etiology* | *Site* | *Main Symptoms* | *Prognosis* | *Treatment in Order of Value* |
|---|---|---|---|---|
| 3 Degenerating diseases—6% (rare) | Most in large vessels | Same at site of disease only | Poor | Restoration rare resection obliteration |

of the placenta, prenatal anoxia and pressure therefore all may be a cause of mental retardation To this must be added the tendency to over-sedate and anesthetize the mothers during delivery If the mother has a systemic disease, the problem of abnormal oxygen intake is greater The use of posterior pituitary extracts to speed up labor, so common today, results in uterine contractions which interfere with fetal circulation[12,14] Clifford[7,8] has pointed out that there is a tendency by the fetus to aspirate amniotic fluid when in an anoxemic state Denny-Brown and Russell[13] showed the concussion effect of a traumatic delivery Abnormal cord constrictions, as pointed out by Eastman[14] and Ehrenfest,[15] require a quick delivery. Increased instrumentation (forceps) also is a cause

As a result of these various causes, there is a cortical atrophy in a higher percentage of children Cerebral blood flow studies in such children showed a decrease from the normal of 72 cc per 100 grams of brain tissue to approximately 40 cc This same concept and principle as to avascularity of the brain has been applied to patients who have had cerebral vascular accidents and hemiplegia

In September 1950, Dr Beck and his associates presented his operation of cerebral revascularization[46] He anastomosed the carotid artery to the jugular vein in a lateral method with ligation and division of the proximal end of the vein This operation was based on the concept that there might be viable brain cells "enmeshed in a gliotic process and that these cells could be improved by increasing the blood supply to the brain " Beck reported on 125 operations[45] He selected patients with a localized or generalized gliosis of the brain as shown by a reduced cerebral blood flow or metabolism

*Theory* —The drainage from the cerebrum is through the right internal jugular vein and from the deep portions of the brain to the left jugular vein Tracers injected through the right internal jugular vein filled the sagittal sinus and the deep circulation The possibility of rupture of the veins under arterial pressure was entertained but dismissed because of the hypertrophy of the intima of the vein showed by Beck[2] and Wolff[68] The hypertrophy of the part after an arteriovenous fistula was considered significant as evidence of increased vascularity Cerebral blood flow was shown to be markedly reduced in patients with organic disease It was thus assumed that if more oxygen could be supplied, cells not functioning because of this lack of oxygen supply could be made active The same theory is the basis for performing the operation on patients who have had hemiplegia

*Technic* —The skin incision is made in a transverse manner at the level of the thyroid cartilage The carotid artery at its bifurcation and the jugular vein are dissected free. All the branches of the jugular vein are ligated The external carotid artery is liberated and its branches ligated Beck originally used the common carotid artery but changed to the external carotid to avoid the occasional cerebral complication It is anastomosed to the cardiac end of the jugular vein by 5-0 Deknatel sutures These sutures are placed in a continuous over and over suture and the fistula is made 3½ to 4 mm in size The distal end of the jugular vein is ligated and divided

*Results*.—These patients have not been followed long enough to give a true picture of the results. The operation is simple and attendant with a minimal number of complications. The evaluation of the results remains the most difficult problem. When a mentally retarded individual improves is a mute point. There have been enough spectacular results to make this operation appear feasible if one can make the correct selection of patients. At the present time, the operation is under critical evaluation by its originators. It appears that the possibility of amelioration of symptoms by this operation is feasible in selected patients. In our four patients so treated improvement occurred in all. The part this operation played in such improvement was difficult to evaluate. The operation should be advised only after due consideration of all the factors involved.

4. *Shunts for Cardiac Pulmonary Edema*.—These operations are discussed under this heading in acquired cardiac disease. (See pages 120 to 121.)

## SUMMARY

In our series of 126 operations on 87 patients with arteriovenous aneurysms the treatment was as follows:

1. Excision of sac and/or veins with end to end anastomosis of the artery. Ideal treatment—applicable in this series in 6 per cent.
2. Excision of sac and/or veins with repair of artery defect (4 per cent). Excellent in recent traumatic injuries and young individuals.
3. Excision of aneurysm: use of analogous vein graft (2 per cent). Excision of aneurysm: use of homologous graft.
4. Multiple ligations and excision of arteriovenous mass (78 per cent).
5. Obliteration with muscle implant (7 per cent). *Applicable where excision is impossible.*
6. Chemical obliteration (3 per cent).
7. Therapeutic uses of this method have been detailed.

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Chapter

22

# AORTIC AND ARTERIAL EMBOLISM

ANY foreign or abnormal particle circulating in the blood may be an embolus and cause an embolism when it lodges in a vessel too small to permit its further passage

**Definition**.—An embolism is a sudden, shocking, usually complete closing of a previously patent major artery by the lodgment of a clot or foreign material (embolus) arising in another part of the circulatory system Usually such an embolus originates on the valves or in the wall or appendages of the heart

**Etiology.**—Embolism occurs most often in patients with left-sided heart disease of the rheumatic type who are fibrillating (96 per cent) Twenty-five per cent of those who die of cardiac disease have an embolism at some stage in their heart disease There is usually some added insult to the system, such as a surgical operation, an obstetrical delivery, or a coronary occlusion Auricular fibrillation is the exciting cause in nearly all instances A change in rhythm to auricular fibrillation occurs in 25 per cent of patients with chronic rheumatic heart disease [38] Mural thrombi are found in one or more chambers of the heart in 13 to 56 per cent of autopsies on patients with chronic rheumatic heart disease [12,15] Age increases the incidence [11,16] One of every four patients treated for mitral stenosis by commissurotomy has auricular or appendage clots [2]

Occasionally, the embolism may arise from the right side of the heart This is paradoxical and requires the presence of a patent foramen ovale It was first reported by Cohnheim in 1877 [7] In more rare instances, the embolus arises from the peripheral arterial system

A patient with sclerotic or other occlusive arterial disease may have a clot or plaque break loose as a result of disease or trauma This loosened plaque may be carried and lodged at a bifurcation of the arterial tree where the circulation is then shut off This occurs much more often than is suspected (19 3 per cent) [13] Trauma causing a local arterial thrombosis may be the source of a later embolism if this thrombosis loosens or liquefies, usually on the seventh to fourteenth day Fat and air embolism are different entities but may occur

**Symptoms.**—*Pain* is an early and important symptom This is usually sudden and may be accompanied by *shock* The pain is located at the site of the embolism and distal to it Pain in any arterial closure is severe, as exemplified by the angina of coronary occlusion Shock may be mild or severe depending on the site of the embolism When the lodgment occurs

at the bifurcation of the aorta the shock may be sufficient to cause unconsciousness and sudden death

There is *tenderness* which is maximal at the point of lodgment and follows the course of the involved artery There may be *muscle cramps* as the part is deprived of blood, especially if the part is moved Where a major vessel is involved *paralysis* occurs distal to the embolism The patient may fall or be unable to walk The *reflexes* will be *absent*

The *temperature* of the part changes It becomes cold and later it may become warm temporarily due to vein blood engorgement to be followed again by coldness

*Color changes* occur at once With the occlusion the blood supply to the limb through that vessel ceases and with reflex vasospasm all vessels cease functioning The limb becomes marble-white in color After a time venous engorgement occurs with blotchiness and cyanosis which eventually becomes more blue in color If the process is not relieved by operation or adequate collateral circulation a beginning demarcation and later gangrene will appear There is usually a fading from the black to a mottled blue to a reddish color and then to a normal pink which may extend to some degree below the site of the embolism

*Venous engorgement* occurs with the color changes mentioned Subsequent venous thrombosis occurs due to venous stasis Mottling is a serious prognostic sign *Blebs* and *skin breaks* may follow this engorgement with tissue death

The *arterial pulsations* theoretically are absent distal to the point of embolism In the early stages they may be so forceful above the embolic area that this force is transmitted along the vessel wall This forceful pulsation above the clot disappears as thrombosis develops distal to the embolus

*Oscillometric readings* usually will be zero distal to the embolus If the collateral circulation is adequate the oscillometric readings may be present but restricted but will not be equal to those prior to embolism or to those in the opposite extremity The blood pressure changes are similar to the oscillometric readings

These symptoms vary in degree depending upon the site of the embolism, the completeness of occlusion the collateral circulation reaction, and the existence of spasm When the embolism occurs at the bifurcation of the aorta the leg symptoms will be bilateral. The symptoms may be mild or more rarely absent if the vessel is only partially occluded or at a site where collateral circulation is adequate In the upper extremity gangrene rarely develops because of more adequate collateral vessels

**Pathogenesis and Pathology** —In rheumatic heart disease vegetation and clots develop in the auricle and auricular appendage or in the ventricle wall These propagate to become mural thrombi With the irregular contractions of auricular fibrillation a portion of the clot may break off and be transmitted to the peripheral arterial system through the left ventricle The added strain of a surgical operation obstetrical delivery or heart attack may be the exciting cause

Showers of emboli or a single one may be displaced These vegetations or clots go through the arterial system and lodge at a point of narrowing

i e, a bifurcation of an artery Most of the emboli (50 per cent) are arrested at the junction of the femoral and femoral profunda arteries Only one in eight occurs in the upper extremity

When these emboli lodge, a local reaction with edema starts in the intima The force of the blood behind the embolism increases the intimal wall pressure and accelerates the wall changes

In over one-half of the cases, the blood distal to the embolus becomes a soft clot This may involve the entire distal arterial tree There may be no arterial thrombosis distal to the clots, the blood continuing to the venous side due to the spasm factor and partly due to the transmission of the pulsations beyond the embolus.

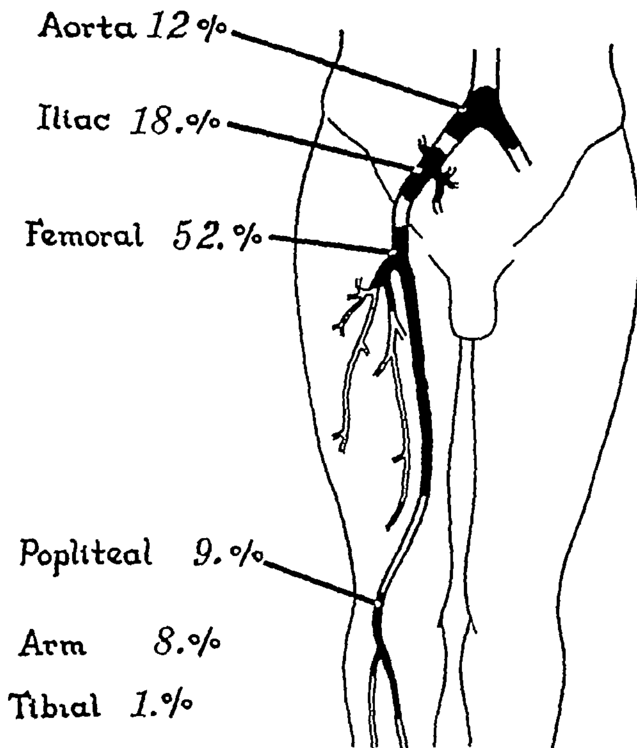



FIG 142 —Sites at which peripheral embolisms lodge in relative percentages (Pratt, courtesy of Am J Surg, amended )

The embolus itself on examination may be only fibrotic tissue or may show collections of partly disintegrated blood cells Papillary projections and vegetation-like growths with out-pouchings may be present Most of the emboli have a bullet appearance, some with a comet-like tail

When the embolus lodges, *spasm* occurs in all cases and affects not only the vessel involved but the collateral circulation This spasm is Nature's protective mechanism to aid in emptying the blood vessel distal to the embolus so that the vessel will not be thrombosed In such vessels the circulation most often can be re-established

In the capillary bed the arterial pressure drops Spasm empties the vessels but then with relaxation the venous blood leaks back into the capillary bed Venous thrombosis then follows

The pathologic picture in embolism is unlike that after simple ligation of a major artery This difference is due to the spasm and the thrombosis in

the rest of the arterial tree Ligation of the first part of the subclavian artery for example will not result in gangrene as reported by Halstead[14] in 1924 Only a small per cent of proximal major artery ligations are followed by gangrene In a simple ligation the collateral circulation often takes up the load while in embolism shock and the cardiac status make thrombosis more likely

**Diagnosis** —The diagnosis of arterial embolism is not difficult It can be made on the history alone The usual sequence is that of a patient with rheumatic heart disease with auricular fibrillation who develops suddenly a cold pulseless extremity, and this is pathognomonic

*The Six "P's of Arterial Occlusion* —The alliterative "six P's of arterial embolism diagnosis are worthy of mention These are pain paralysis, paleness, pulselessness, paresthesia and prostration In the absence of effective therapy all will be present

**Differential Diagnosis** —Arterial thrombosis and, at times thrombitis (thrombophlebitis) may simulate embolism and require differentiation

Arterial thrombosis arises in the artery due to trauma or to a degenerative disease Heart disease and auricular fibrillation are not necessarily present in arterial thrombosis but the presence of arteriosclerosis in other vessels is a significant point. Thrombitis (thrombophlebitis) at times has been confused with embolism due to the reflex arteriospasm which accompanies some vein thrombosis In thrombophlebitis the leg is warmer, more cyanotic and edematous While the major artery pulsation may be absent this is a temporary status due to spasm It can be demonstrated by relaxing the spasm Paralysis and the other differential points of embolism listed under symptoms are important

The differential diagnosis of these three lesions is summarized under Acute Arterial Thrombosis on page 3o7

**Prognosis.**—The prognosis in embolism is never good Figures vary greatly and depend upon the physical status of the patient particularly his heart the site of embolus location and the treatment

*Aorta* —Of Andrus's 10 patients 70 per cent died and the other 30 per cent developed gangrene of one leg[1] Of Haimovici's 28 19 died or developed gangrene[12] One-half of our 12 aortic embolectomies died

*Iliac* —Prognosis in this group will be slightly better only 4 of 53 in Haimovici's[12] series expired although 32 had gangrene.

*Femoral* —Prognosis in this area is better The figures vary from 20 to 40 per cent gangrene and approximately the same number die Fourteen of our 42 femoral embolectomies died

*Popliteal and Distal Arteries* —Our experience with embolism in this area has been poor despite all methods of treatment Other authors have reported encouraging results but unless the diagnosis was confirmed surgically it could be questioned at least on the basis of our Clinic's observations A clinical pathologic study of 50 emboli arising from the heart showed that 40 to 50 per cent of the thrombi originate in the left auricular appendage[17]

**Treatment.**—There has been dispute as to the advantage of medical or surgical therapy and proponents for both types have been forceful There is no doubt that indications for both types of treatment exist, and in many

the management should include both. The surgeon who arbitrarily operates on a decompensated and obviously moribund patient does a disservice. Such a patient's primary problem is his heart, and if this can be corrected medically while conservative measures are instituted for the embolism, the patient has a chance for survival. The internist who keeps a younger patient on a medical regimen while gangrene of one or both limbs develops also is culpable, especially if the cardiac status is fairly good. Further experience has caused us to modify in practice while retaining in principle the dictum that the treatment of arterial embolism should be surgical as soon as the diagnosis is made.

When the arterial embolism is diagnosed, the patient's status should be evaluated at once. During this time of necessary delay, sympathetic blocks and vasodilating drugs can be given. If at the end of two to four hours there is no improvement, embolectomy should be considered and rejected only if the general status precludes any operation. In this respect one should consider the prognosis without operation carefully, and if such is nil, operation should be elected. Patients with embolism are poor risks for life because of their cardiac status. Forty per cent of Haimovici's 330 collected embolisms of all types died or had gangrene.[13] Only 1 in 4 will be alive in five years. Embolectomy is only for restoration of the arterial circulation in the affected limb or limbs. If the operation accomplishes this purpose, it is successful. Without embolectomy, most of these patients die in a few days. In my series of 52 embolectomies, the circulation has been restored in 60 per cent of the instances. In others, an amputation has been followed by months or years of life.

The poor general prognosis is not a contraindication for the operation. While only 1 in 4 patients with carcinoma of the breast will be alive in five years, operation is not refused to this group. In some, other emboli may develop. In others, control of the cardiac status with the anticoagulants may keep the patient alive for many years.

*Surgical Treatment.*—Delay in the institution of surgical treatment is the reason for most failures. There are many reasons for this delay. The diagnosis for a time may be doubtful. Waiting for a consultant may lose many valuable hours. The usual explanations and discussion among the patient's relatives as to therapy interfere with early operation.

If surgical intervention is selected, there should be no efforts to overcome the spasm factor, for this spasm is a protective mechanism, as has been described. (See page 440.)

The surgical treatment of embolism is of recent origin. The early operations were unsuccessful. S. Sabanejew[34] explored the first patient for an embolus in 1895 but did not locate the clot. Lord Moynihan,[29] in 1897, removed an embolus from the popliteal artery, but the patient later succumbed. Doberauer[9] and Stewart,[36,37] in 1907, resected emboli, but thrombosis followed and the operations failed. Trendelenburg[39] tried unsuccessfully to resect an embolus in the same year. Carrel,[6] Murphy,[25] Leriche,[22] Schiassi,[30] and Proust[33] all tried the operation in 1908 and 1910 without success.

A nearly successful embolectomy was performed by Labey[20] in 1911, with Key's[18,19] encouraging work following thereafter.

The Scandinavian surgeons had performed 150 embolectomies up to 1928 but their work was practically unnoticed in the United States and England where under 20 such operations were attempted Since that time many embolectomies have been performed—over 400 in the Scandinavian countries alone and many hundreds in the United States with approximately 1 in 4 surviving five years

**Operative Site** —The ideal operative exposure in embolectomy has been disputed, particularly when the clot is in the aorta or iliac area This is still a question in many surgeons minds.

Many surgeons believe that a direct exposure of the vessel at the embolus site is of primary importance to the surgical success [31,40] In the successful embolectomies reported by McClure and Harkins [illegible] 10 were done through the extra or intraperitoneal operation and 7 by a retrograde approach

The direct approach is advocated for the removal of all emboli except those at the bifurcation of the aorta or the iliac arteries

The disadvantages of the direct approach to these latter vessels are

(1) Considerably more operative trauma time, and anesthesia are required Thus a patient whose general condition is very poor has to have a major operation at a time when he can withstand it least

(2) To adequately expose this part of the aorta or iliac artery either transperitoneally or extraperitoneally packing and retraction of the peritoneal contents is necessary A diseased heart is further embarrassed by pressure against the diaphragm

(3) To mobilize the aorta and the common iliac vessels is a major undertaking and not without risk of secondary thrombosis or hemorrhage

(4) A clot may slip away during such manipulation secondarily lodging at a much more common and dangerous site either at the femoral profunda site or in the popliteal area on either side In addition the distal clot beyond the embolus often cannot be removed

(5) Hemorrhage in the opening of the aorta is always difficult to control despite any type of tourniquet or manual pressure applied to the major vessels.

(6) In applying the necessary pressure thrombosis may result from a fracture of the intima or the entire wall of the large vessel

(7) The suturing of the artery in this area is difficult technically

(8) If the embolus is not readily demonstrable the difficulty of attempting to aspirate it by suction or with some instrument may be insurmountable

(9) These emboli can be removed retrogradely and successfully in a higher percentage of cases without the direct approach Surgical trauma is minimal in the femoral area compared to the direct approach to the aorta.

(10) The diagnosis of embolism at the bifurcation of the aorta or in the iliac arteries may be erroneous. The clot more often is at the junction of the femoral or femoral profunda arteries where approximately 60 per cent of the embolisms lodge Embolism at the femoral area causes proximal spasm In addition venous congestion will mottle the abdomen and even the chest wall

**Retrograde Embolectomy.**—*Advantages* of the retrograde method are (1) The surgical approach is simple, it can be performed under a local anesthesia and is not attendant by the same operative trauma or shock as is the direct approach

(2) Approximately 60 per cent of all peripheral embolisms will be found at the femoral-femoral profunda site Where the embolus is at the site of the femoral or femoral profunda arteries, it can be removed without instrumentation

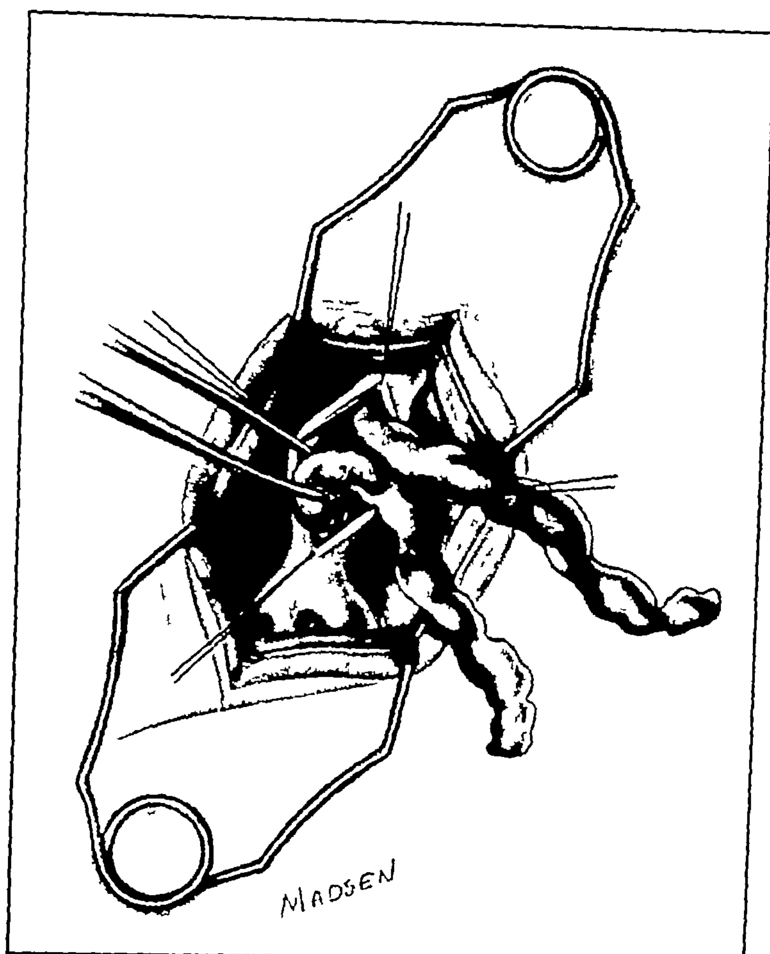



FIG 143 —Embolectomy of femoral artery Removal of accompanying thrombus (Pratt, courtesy of Am J Surg )

(3) The artery is subcutaneously placed and can be lifted from its bed without trauma

(4) The procedure can be performed on both sides at the same time in case the embolus is above this point without loss of time or further shock

(5) A small rubber catheter, with suction applied, in most instances will release an embolus lodged higher

(6) An adherent clot from above can be removed by milking it manually or by the introduction of some type of corkscrew (See page 446 )

(7) In over one-half of the patients who have an embolism there is an attendant thrombosis to the embolism distal to the point of embolism. If this thrombosis is left after the embolus has been removed, the procedure

must fail With the retrograde method this distal thrombosis can be extracted as shown in Figure 143 In many instances, the thrombus will come out in one piece and completely free the distal arterial tree. Adequate removal of a distal thrombosis is not possible when an approach is made through or behind the abdomen

*Anatomy* —The femoral artery is superficially placed in this area and must be approached carefully The artery directly overlies the femoral vein rather than being lateral to it as is described in most anatomy books These two vessels are in a common sheath in the femoral triangle This triangle (Scarpa s triangle trigonum femorale) corresponds to the depression in the thigh below the groin It is formed medially by the adductor longus muscle and laterally by the sartorius muscle It has its apex distally As a floor it has portions of the psoas major, iliacus and the pectineus muscles

The femoral sheath (crural sheath) is an extension of the abdominal fascia, made from the iliac fascia behind and the transversalis fascia in front of the vessels This fascia fuses with the covering of the vessels about 4 cm below the inguinal ligament Anatomically the artery and vein are adherent Separation of the two must be made carefully to avoid injury to the vein Where there has been an antecedent vein inflammation the adhesion of the two vessels will be greater

*Operative Technic* —*Incision* —The incision *for the removal of all emboli from the bifurcation of the aorta to the femoral area* is made vertically just below the inguinal ligament for a distance of three inches This incision is made 1 inch medial to the spine of the pubis The superficial and deep fascia are divided and the femoral artery is exposed This artery and the femoral profunda artery are dissected free in an atraumatic fashion

When the femoral and femoral profunda arteries are exposed rubber bands are passed around the artery to gain control proximal and distal to the embolus The rubber bands are run through a piece of rubber tube which is clamped to avoid pressure of clamps on the vessel

The Bethune-type of tourniquet clamp as recommended by Linton[23] and as used in lobectomies also is an excellent tourniquet. The vessel is occluded over a wide surface somewhat as if pressed between two thumbs in contradistinction to the more traumatic occlusion by a clamp The use of this type of tourniquet is advocated where any large vessel temporarily has to be occluded

After the femoral artery is exposed it is incised longitudinally The embolus or its attached thrombus usually herniates through the incision

The thrombus is teased out from the distal end of the artery first This soft clot will slide out readily and can be lifted with a smooth forceps At times it will come out in one piece and a cast of the entire distal arterial tree may be obtained *This part of the operation is important* as the subsequent circulation depends on the open distal arterial tree. If difficulty is encountered suction or milking may be required The proximal end of the clot is then extracted The thrombus often breaks off at the embolus junction depending on how long it has been lodged

The embolus itself then must be removed. A neurologic type suction tip or suction on a catheter may move it if it is of recent lodgment If

many hours have elapsed since the embolus occurred, it may be tightly adherent to the wall Adhesions are caused by the constriction of the larger embolus into the smaller lumens at the artery bifurcation and the pressure behind the embolus An inflammatory process with edema of the wall is set up at the embolus lodgment point in a few hours' time If the embolus cannot be freed with suction, a smooth forceps may pry it free We use a smooth, curved, modified gallstone clamp for this purpose

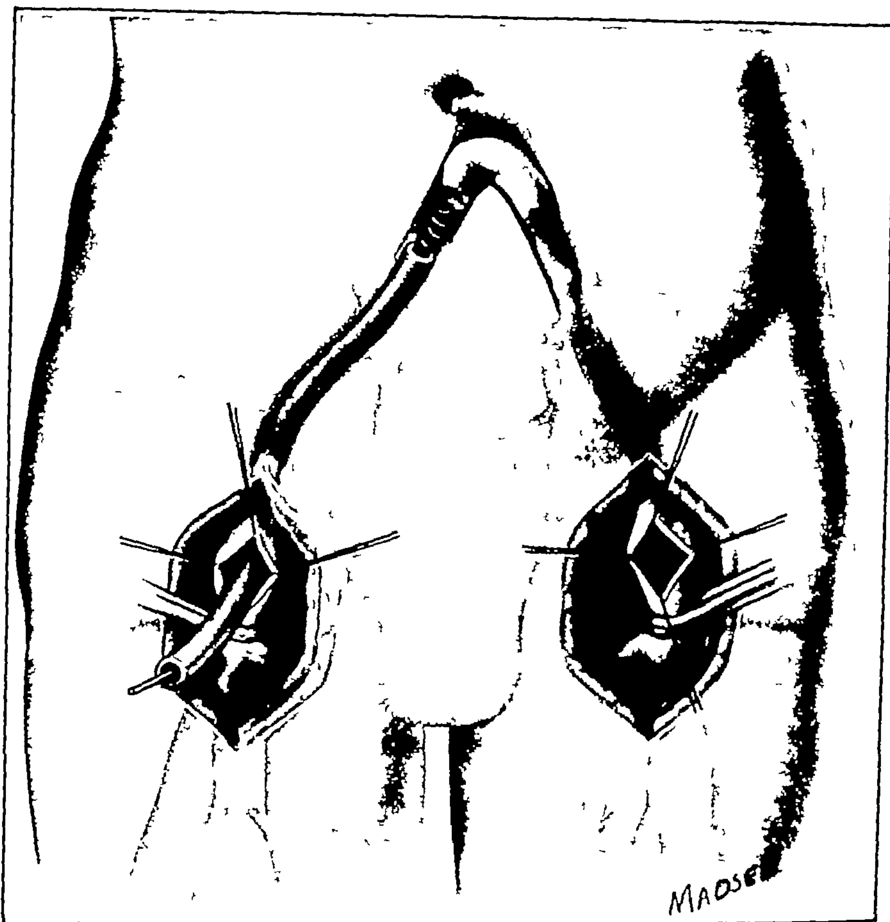



FIG 144 —Retrograde approach to embolism at bifurcation of aorta Both femoral arteries are opened and are controlled by tapes A corkscrew instrument made by winding a silver wire on a probe has been introduced into the embolus This wire is enclosed in a catheter to protect the intima This corkscrew is then turned through the embolus and withdrawn (Pratt, courtesy of Am J Surg)

If this is ineffectual, a corkscrew instrument is constructed This is made by winding a large-sized silver wire on a probe The sharp point of the wire is turned over to protect the artery intima This wire corkscrew is then introduced into the artery covered over with a small catheter When the embolus is reached, the wire is pushed forward and turned through the embolus as through a cork. When the wire has penetrated the embolus, fresh arterial blood pumps out along the wire The wire is then pulled out without turning The embolus will come out like a cork on the wire curls Care must be taken to prevent any clot passing distal to the artery opening at this stage The corkscrew may have to be passed again. At times a larger caliber wire is needed to free it

In other instances, the hand may be passed up beneath the artery and the vessel manually "milked" to free the obstruction The operator's hand has been passed retroperitoneally up to the bifurcation of the aorta to successfully "milk" the adherent embolus

Considerable ingenuity may be required to free the artery completely Some of the instruments we have used at this stage include biopsy forceps bronchoscopic probes, forceps, foreign body removers spring probes gallbladder probes and clamps vein strippers uterine sounds and all types of suction and probes made of various wires at the operation time

*Aorta bifurcation* and *iliac artery embolisms* are treated in a similar way the clot first being removed retrogradely as described

When the embolus is freed there will be a sudden burst of fresh arterial blood This will continue forcibly with each systole A few squirts of blood may be permitted to be certain that the artery is clear of clot The *bleeding* is then controlled by mild traction on the rubber band and the stay sutures previously placed Free bleeding from the distal end of the artery indicates an adequate collateral circulation around the artery opening and improves the prognosis

The artery is then washed out with heparin solution and 500 units of heparin may be left in the artery An intraarterial injection of 800 units of heparin in saline after the arterial suturing is complete is suggested [11] Regional heparinization by tubing inserted just above the suture line for as long as three days has been suggested [10] It can be sutured into a branch Local hemorrhage may be a problem

The artery wall is then *sutured* with a continuous running suture of 5-0 silk on an atraumatic needle This closure is best obtained by approximation of the external artery coats only as this adequately provides the arterial repair needed If required the artery can be closed by an everting mattress suture but this decreases the available lumen materially As there is already considerable spasm this may interfere with adequate blood passage at the suture point In our experience the external coat closure is more satisfactory

The suturing technically is simple The walls need only to be brought into approximation as the arterial blood flow tends to pass directly down the artery once it is closed This occurs because the wall pressure at any point in a vessel wall containing a moving fluid is inversely proportional to the rate of flow The rate of flow in the artery being rapid the wall pressure is low No reinforcement of the suture line is needed The subcutaneous layers then are closed with fine catgut to prevent any silk sinuses and the skin is sutured

**Direct Approach to the Aorta or Iliac Artery for Embolectomy** —Spinal anesthesia is advocated for the direct approach of the aorta or iliac arteries either intra or extraperitoneally This anesthetic agent permits the relaxation necessary for exposure and has proven to be the safest Although this approach is not advocated in general for the reasons outlined on page 443 the technic is given here There may be occasions when it is necessary to approach directly the involved vessel

(1) *Transperitoneal Approach to Aorta or Iliac Artery* —A right left or midline incision is made at the level of the umbilicus The abdomen is

opened and the viscera packed into the upper abdomen aided by the Trendelenberg position The cecum and sigmoid are displaced to either side with packs The retroperitoneum is opened at the aortic or iliac artery level The involved vessel is mobilized and rubber tapes are passed around it Manual pressure may replace the tapes. The vessel is opened in its long dimension

Considerable difficulty may be encountered in removing the embolus and its thrombus and in controlling the bleeding Rubber catheters make excellent tourniquets or the Bethune-type of lobectomy clamp may be used at this stage Suturing of the artery must include the entire wall in an everting type of mattress suture, the suture being an arterial 4-0 silk on atraumatic needles

It should be remembered that bleeding tends to stop when the constriction of the large vessel is removed, thus increasing the rate of flow and decreasing the wall pressure at the incision point

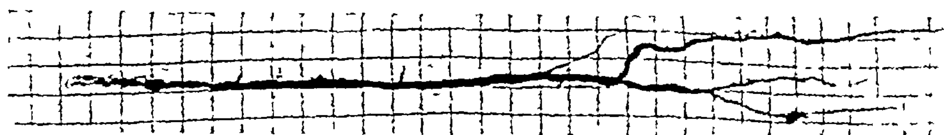


FIG 145 —Embolus in iliac artery with thrombus of arterial tree removed in one piece Note clot in branches Referred by Dr C Poindexter (Pratt, courtesy of Am J Surg )

(2) *Retroperitoneal Approach to the Aorta* —This is the same approach as is used for the exposure of the lumbar sympathetic chain described on page 507 The position of the patient is most important The operative side should be raised to permit the viscera to fall away from the surgeon but a direct lateral position is not indicated A moderate-sized sandbag under the hip, combined with a table break will give the desired exposure A transverse incision is made 2 inches above the crest of the ilium and over the external oblique muscle on the left side The external oblique and internal oblique and transversalis muscles are divided in the line of their fibers The transversalis fascia should be separated well lateral to the rectus muscle to avoid injuring the peritoneum to which it is adherent medially

The peritoneum and its contents are retracted to the mid-line If necessary this maneuver is aided by raising the kidney rest of the table and tilting the table to the opposite side The ureter retracts with the posterior peritoneum The aorta is dissected and rubber tapes passed around it A small McBurney retractor is an excellent blunt ligature carrier for this work The embolectomy and the closure then proceed as described before See page 447

(3) *Retroperitoneal Approach to the Iliac Artery* —In this approach, the incision is a lateral one from the anterior superior spine of the ilium on the involved side for 3 inches running in the direction of a McBurney appendix incision The muscles are split as before (see above) The mobilization of the vessel and the embolectomy proceed as described on page 447

In all such transperitoneal or retroperitoneal approaches to the large vessels, a lumbar sympathectomy should be performed at the same time

The sympathectomy requires little extra time and may save subsequent spasm complications

Closure of the wounds is accomplished with interrupted steel wire or other sutures buried in the fascia

The aftercare is similar to that outlined following retrograde embolectomy See below

**Postoperative Care** —(1) **Anticoagulants** —Anticoagulant therapy is begun at once with heparin and the usual doses of the oral anticoagulants See chapter on Antithrombotic Therapy page 651 An elevated prothrombin level indicates heparin can be discontinued Heparin is best given subcutaneously

If heparin is used the technic of administration should follow that described in the chapter on Antithrombotic Therapy page 651 The oral anticoagulants also should be used as described in the same chapter Prophylactically, this drug may be continued indefinitely

(2) **Interruption of the Sympathetic Innervation.**—Spasm is a factor and may continue even after the embolus has been removed By novocain blocks of the sympathetics the spasm may be relieved and these blocks may be repeated as often as is necessary A polythene tube can be left *in situ* to avoid repeated needle punctures

The sympathetic system may be interrupted permanently by operation if the results of the local blocks are beneficial but too temporary Sympathectomy may be required at a later date to aid the deficient circulation

Chemical sympathectomy may be achieved with any of the following (*a*) tetraethylammonium chloride which has the trade name Etamon (*b*) dibenzyl beta chlorethyl amine hydrochloride known as 'Dibenamine (*c*) 2 benzyl-4,5 imidazoline hydrochloride, which is under the trade name Priscoline ' Their value and dangers are discussed on pages 490 to 494, Sympathectomy

These drugs may help if the usual sympathetic blocks are contraindicated Intravenous novocain or ether also may help temporarily

(3) **The Care of the Limb** —After an embolectomy the circulation in the limb will be in jeopardy for some time. This may be due to spasm There may have been some thrombosis distal to the emboli not removable at the operation which may interfere with the circulation Thrombosis at the operative site may occur

The part should be protected against injury by handling it with extreme care and wrapping it in candy cotton A slightly dependent position is advocated unless swelling occurs

The limb should be exposed in a bed cradle at room temperature which provides the best medium for a limb s recovery

(4) **Drugs** —Whiskey and aspirin are effective mild vasodilators Papaverine hydrochloride in a dosage of 60 mg and eupaverine may be beneficial. Spasmalgin which contains atropine pantopon and papaverine may be given intravenously or intramuscularly every two hours if needed Papaverine as the only treatment resulted in 17 per cent deaths and 14 per cent gangrene in Haimovici s series[12] and 20 per cent in Denk s [8] The newer sympatholytic or adrenolytic drugs should be used with caution because of the hypotension which may result

(5) **Cardiac Status.**—Digitalization should be carried out in these patients There is no proof that the stronger, slower muscle contractions liberate more emboli The fast irregular beats of auricular fibrillation, on the other hand, do cause emboli to break loose

(6) **Refrigeration.**—Cooling of the part decreases the demand for oxygen in the experimental animal There has been no proof that similar cooling reduces the necessity for blood supply, particularly in the limb with a deficient circulation The use of cooling cannot be advocated as primary therapy by the author Other writers are in agreement [42] The limb may be cooled if the circulation appears lost Some limbs have recovered thereafter. This may have occurred because the cooling reduced inflammation or merely for coincidental reasons

**Other Therapy.—Anticoagulants.**—Some patients with rheumatic heart disease have a tendency to multiple embolisms. For example, we have one patient who has had six separate embolic phenomena that were definitely diagnosed and probably many others not clinically evident. This patient weighs over 300 pounds and operation is not possible She has been treated successfully at home with anticoagulants since their introduction

Ambulatory antithrombotic therapy has been used safely for years That such a program is effective is attested by the fact that we have used it successfully in our Vascular Clinic at St Vincent's Hospital in New York This has been done despite the fact that the mental level of many of these patients is not too high A pattern of their response to the drug is obtained by daily tests Thereafter, a prothrombin test is run only every two weeks The patients are given instructions to observe themselves for bleeding signs There have been but two hemorrhages and neither of these was serious See chapter on Antithrombotic Therapy, page 651

**Ligation of the Auricular Appendage.**—In some patients who have massive embolisms coming from mural thrombi originating in the auricular appendage, surgically ligating this auricular appendage has been tried Since so many of these clots originate in this area and are harbored there, a mechanical block to their propagation into the arterial circulation can be performed

Madden[27] has reported on two such instances That this appendage can be resected surgically has been proven in the experimental animal and in the multiple operations for mitral stenosis now reported [2,5] (See pages 112 to 114 ) Longmire and Beal,[24,25] Baronofsky[3] and others have now reported on auricular appendage removals About 25 per cent of those operated for mitral stenosis had clots in the auricular appendage [2] This operation is feasible and fairly safe if the patient's physical status is good It should not be used in place of anticoagulant therapy but in conjunction with this treatment If elected the approach to the auricular appendage should be the same as that described in the operation for mitral stenosis (See pages 112 to 114.) Great care should be utilized to prevent dislodging the clot at the operation When the purse string suture has been placed, the appendage is amputated Several forceful contractions of the heart may be permitted to force clots out through the cut appendage A temporary occlusion of the right innominate and the left carotid artery is advocated during this procedure to prevent the 5 to 10 per cent cerebral emboli

reported after commissurotomy After the clot is removed the auricular appendage stump should be over-sewn with continuous surgical silk size 00 on an atraumatic needle The author s appendage needle is suggested as a helpful adjunct The indications for this operation exist It will be used in those uncontrolled by anticoagulants or where repeated embolisms make the risk of operation less than that of the repeated assaults on the arterial system The operation should be considered if the antithrombotic drugs are not indicated The good results achieved with this long antithrombotic therapy have relegated the operation with its risk to a last resort It is not recommended that this procedure be followed unless coincidental commissurotomy is contemplated

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Chapter

# 23

# SURGICAL TREATMENT OF OCCLUSION OF A MAJOR ARTERY

*Occlusion Due to Trauma Embolism Disease Infection, Pressure Cold*

## MEDICAL MANAGEMENT OF ACUTE ARTERIAL OCCLUSION

THE surgical management of acute arterial occlusion is described under the various diseases It *is summarized in this chapter* so *that* when an acute arterial occlusion occurs the details for its surgical management are available in one place The principles applicable to injuries to arteries as occurs in accidents the patient s work or in war are in the chapter on Traumatic Injuries to Arteries pages 321 to 351

The life or death of any part of the body depends upon its blood supply There is no greater emergency in surgery than when the blood supply to any part is shut off The part thus supplied by the blood vessel dies unless the circulation is re-established or collateral blood vessels are able to carry the arterial blood If the part of the body is necessary *for life such as* the brain the heart muscle the kidneys the lungs or other vital structures the patient dies dependent upon the success or failure to re-establish the continuity of the blood supply When a small artery is occluded, a similar serious problem may arise by the action of spasm on major vessels

When a major blood vessel becomes occluded suddenly and acutely the physiologic response is not the same as when it is gradually occluded In the gradual occlusion collateral vessels are developed as the major vessels slowly close In the acute occlusion a collateral circulation has not been developed

Spasm plays an important part in any sudden occlusion When a major blood vessel closes acutely the rest of that vessel and the collateral vessels go into arterial spasm This is a physiologic spasm and is Nature s effort to preserve the circulation to the part (see Fig 140)

Spasm produces shock which reduces hemorrhage. The spasm also empties the blood vessel and the collateral vessels involved This prevents blood from remaining in these vessels and clotting thus making the restoration of the blood supply impossible This spasm also affects veins.

When some circulation is restored this spasm usually relaxes For this reason an early effort to relax this spasm may permit some blood to enter the collateral vessels or the main vessel and become static and clot before adequate circulation is available. Whether the spasm is on the basis of a reflex neurogenic syndrome or is accomplished by the release of some

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Chapter

# 23

## SURGICAL TREATMENT OF OCCLUSION OF A MAJOR ARTERY

*Occlusion Due to Trauma, Embolism, Disease, Infection, Pressure, Cold*

### MEDICAL MANAGEMENT OF ACUTE ARTERIAL OCCLUSION

The surgical management of acute arterial occlusion is described under the various diseases. It is summarized in this chapter so that when an acute arterial occlusion occurs the details for its surgical management are available in one place. The principles applicable to injuries to arteries as occurs in accidents, the patient's work or in war are in the chapter on Traumatic Injuries to Arteries, pages 321 to 351.

The life or death of any part of the body depends upon its blood supply. There is no greater emergency in surgery than when the blood supply to any part is shut off. The part thus supplied by the blood vessel dies unless the circulation is re-established or collateral blood vessels are able to carry the arterial blood. If the part of the body is necessary for life, such as the brain, the heart muscle, the kidneys, the lungs, or other vital structures, the patient dies dependent upon the success or failure to re-establish the continuity of the blood supply. When a small artery is occluded, a similar serious problem may arise by the action of spasm on major vessels.

When a major blood vessel becomes occluded suddenly and acutely, the physiologic response is not the same as when it is gradually occluded. In the gradual occlusion, collateral vessels are developed as the major vessels slowly close. In the acute occlusion a collateral circulation has not been developed.

Spasm plays an important part in any sudden occlusion. When a major blood vessel closes acutely, the rest of that vessel and the collateral vessels go into arterial spasm. This is a physiologic spasm and is Nature's effort to preserve the circulation to the part (see Fig. 140).

Spasm produces shock which reduces hemorrhage. The spasm also empties the blood vessel and the collateral vessels involved. This prevents blood from remaining in these vessels and clotting, thus making the restoration of the blood supply impossible. This spasm also affects veins.

When some circulation is restored, this spasm usually relaxes. For this reason an early effort to relax this spasm may permit some blood to enter the collateral vessels or the main vessel and become static and clot before adequate circulation is available. Whether the spasm is on the basis of a reflex neurogenic syndrome or is accomplished by the release of some

spastic secretion into the blood stream, such as adrenalin or spastin, is only of theoretical importance The spastic end-result is an accepted fact

The treatment of acute arterial occlusion will be considered according to the cause

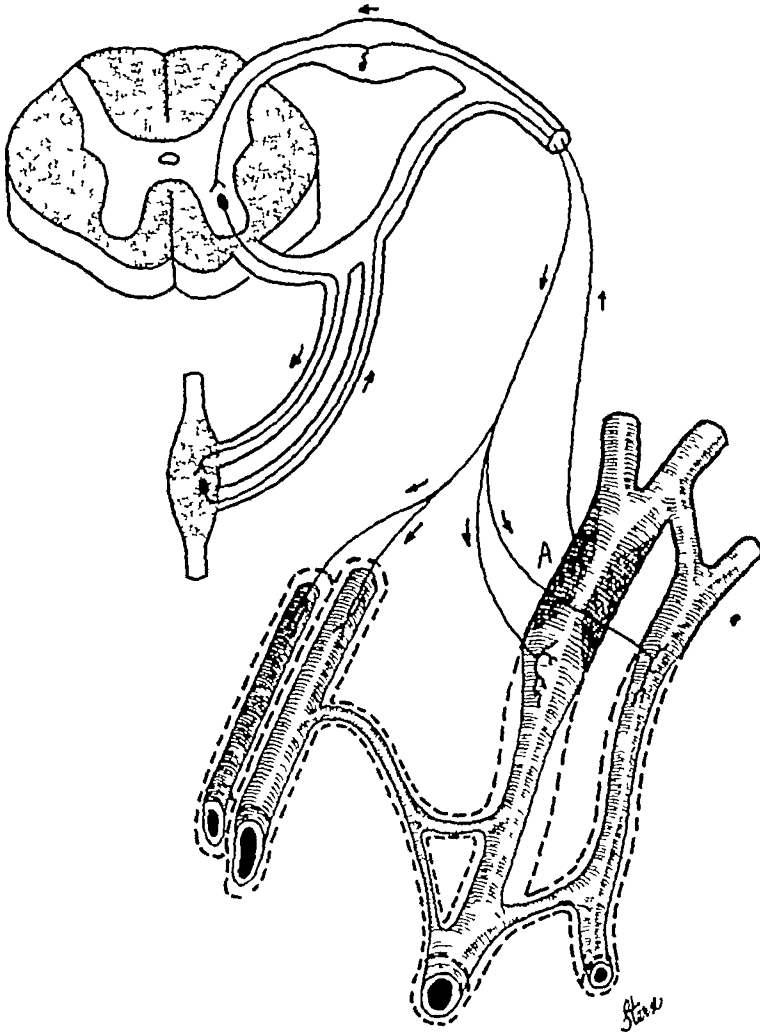



FIG 146 —Reflex angiospasm An obstruction such as a sclerotic plaque at A initiates afferent stimuli Efferent stimuli from the ganglia cause spasm in the effected vessels, collateral vessels and veins The pathway may be neural or chemical

## TRAUMATIC ARTERIAL OCCLUSION

Trauma may cause an arterial laceration or contusion If there is a laceration, there will be marked hemorrhage both externally and internally unless it is controlled by occlusion or spasm Where there is contusion, there may be evidence of loss of circulation to the part

In acute arterial occlusion due to trauma, the pathologic picture is usually a local thrombosis Secondary hemorrhage by pressure in the subcutaneous tissues may cause the same picture

The symptoms of traumatic arterial occlusion depend on the site and the degree of trauma but are always those of arterial closure, with coldness of the part, paleness, absence of pulse and blood pressure distally, anesthesia, and usually paralysis. (See page 330.) Later, there may be some mottling

as the venous blood stagnates and runs back into the capillary bed If not relieved this mottling later becomes an area of demarcation and the end picture is gangrene

A short delay for observation is indicated in all such patients If at the end of a few hours improvement has not occurred the part should be surgically explored The blood vessel should be exposed and proximal as well as distal control of the vessels obtained Distal control of the vessels is necessary as serious bleeding may arise from the lower end of the vessel due to collateral supply

If a large artery is involved *i e* the femoral artery the iliac arteries should be exposed through a separate incision for control The clavicle, sternum or fibula should be removed if necessary to get proximal control If the wound is recent and open the tract should be excised

The site of injury can then be exposed directly all clots debris and slough removed and the wound surgically cleansed in the same way as for any other traumatic wound (See page 331)

**Surgical Management of Lacerations** —Lacerations will be complete or incomplete

(1) *Complete Lacerations* —If the laceration is complete and the patient is seen early, after control of the vessel all clots are removed If an adequate blood flow is obtained the edges of the artery are freshened the blood vessel washed out thoroughly with heparin-saline solution and an arterial repair made in an end to end fashion

If there is *no tension,* this repair can be made with fine sutures with 5-0 silk and fine eye-type needles the sutures being placed only through the adventitia and the muscularis layers but not entering the intima

If there is *tension* this can be overcome by the rerouting of the blood vessel or the flexing of the part The suturing then is best performed by an everting mattress-type of suture this suture going through all of the layers of the walls

Heparin solution can be left inside the blood vessel

Where the vein has been traumatized or injured it is best to divide it at a distance from the wound and open it If a thrombus is present it should be removed The vein then should be divided and ligated

If repair of the artery is not feasible it should be ligated or sutured, if possible just distal to a collateral branch If considerable dissection is necessary with the opening of fascial planes the vessel should be ligated as close to the wound as possible

The usual treatment for any traumatic wound is then applied Dead tissue is excised Penicillin or other antibiotics are given as well as tetanus and gas gangrene antiserums

It should be emphasized that *no traumatic wound should be closed tightly*

(2) *Incomplete Lacerations* —If the laceration is incomplete and the patient is seen early the arterial wound is treated as above If the laceration is small the artery may be repaired with an everting type of suture If the injury has been present for some time a pulsating hematoma or false aneurysm may have developed It may be necessary in these cases to divide the artery and to excise or obliterate a segment of it as in an aneurysm (see page 376) Such therapy is never an immediate

procedure unless there is uncontrollable hemorrhage Time is allowed for collateral circulation to develop and the surgeon must be prepared for a major procedure with adequate assistance and blood supply available

In respect to suturing large vessels, the hydrodynamic law, i e, that the wall pressure at any point in a vessel containing moving liquid is inversely proportional to the rate of flow, applies The wall pressure is low in an artery, as the rate of flow is rapid

Where arteries and veins are both injured, an arteriovenous fistula may develop After six months, an excision of the arteriovenous mass is indicated This time interval may be modified if signs of rupture or circulatory failure distally develop Such arteriovenous connections are always followed by development of collateral circulation within a few months An excision rarely results in gangrene In a few cases, the laceration of the artery may be repaired with excision of the vein (See Arteriovenous Aneurysm, page 417.)

**Surgical Management of Contusions of Arteries.**—Contusions may cause direct or indirect thrombosis The indirect thrombosis may be relieved by removing the blood clot that has developed after the contusion If the circulation has not been restored, the artery should be exposed at the contusion point, control obtained, and the vessel incised in its longitudinal direction Thrombectomy then is performed, similar to that outlined under Arterial Thrombosis and Embolism See pages 361 and 445 Any thrombosis distal to the site of contusion should be removed at the same time and through the same incision.

Medical efforts to re-establish the circulation should be continued until the outcome is decided

## ARTERIAL EMBOLISM

Arterial embolism has been discussed completely in the previous chapter. It is summarized here briefly

The patient who develops an arterial embolus, in general, is one who has left-sided heart disease with auricular fibrillation and usually has had an operation, an obstetrical delivery, an injury, or a coronary heart attack to initiate the embolism

Conservative therapy may be tried for a few hours This includes relaxation of spasm, anticoagulants and general systemic support If not successful, surgical embolectomy must be considered and elected if the general physical status permits operation

Where the aorta or the iliac arteries are involved, it is our practice to explore retrogradely through an incision made at the femoral artery and the femoral profunda junction The artery is opened with a longitudinal incision and the embolus exposed The thrombus distal to the embolus should be removed first, then the embolus is extracted In selected instances, an embolus at a higher level must be exposed directly. While such occasions are rare, it is necessary at times to open the iliac arteries or the aorta Whether the operation is performed trans- or retroperitoneally, the operative mortality will be greater than by the femoral approach The reason for this increased mortality is the necessary greater shock and

manipulation in an already seriously ill individual This problem was discussed on page 445

The methods of removing an embolus have been discussed fully in the previous chapter on Arterial Embolism page 438

If the patient is seen late or the embolectomy fails all conservative measures to stimulate the collateral circulation, including nerve blocks and anticoagulant therapy, are indicated Interruption of the sympathetics and the administration of anticoagulants are indicated also after an embolectomy

## NONTRAUMATIC ARTERIAL THROMBOSIS

Acute arterial occlusion by thrombosis may occur without injury due to infections blood dyscrasias congestive heart failure, and any condition causing a lowered blood pressure or slowed blood flow (See chapter 19 ) Occlusion also may occur in vessels which are already diseased In these cases the lumen of the artery gradually has been reduced Due to undermining or some slight trauma or increased intra-arterial pressure, a section of calcium or a plaque will be loosened and swung across the lumen, like a door to completely occlude it The closure then is an acute one The treatment of the underlying cause is obvious

A thrombectomy is possible

The part that endarterectomy or resection of the vessel with or without a graft may play in selected patients has been discussed on pages 203 to 210 Arteriograms have altered the program in many instances The treatment of acute arterial thrombosis as well as traumatic arterial thrombosis also have been outlined in detail in the chapter on Arterial Thrombosis page 352

We have removed plaques and debris with wires probes corkscrews and suction Bazy's[1] technic of removing all but the outer coat of the artery has a place in many instances.

## INFECTIOUS ARTERIAL OCCLUSION

Acute arterial occlusion may be caused by infection Hemolytic streptococci and *Clostridium perfringens* (*cl welchii*), the gas gangrene bacillus are the most common causes of such occlusion Where the occlusion is caused by an infection there is usually an injury with a portal of entry although the gas gangrene organisms may cause infections secondary to gangrene. Other infectious diseases may cause an acute arterial occlusion By direct invasion of the blood stream the disease may cause changes in the blood supply to produce the occlusion. There may be so much reaction in the part of the body particularly in the extremities that the inflammation venous stasis and edema may close off the arterial supply Typhoid fever, influenza, pneumonia typhus fever and the acute septic infections are common causes Occlusion has been reported in connection with ulcerative colitis In some instances abscesses of the walls of the arteries develop in any or all of the coats. These may be the result of direct extension of a surrounding infection or be embolic or metastatic from another part of the body

When such infections are first seen, conservative therapy may be tried The regimen includes

(1) *Adequate Chemotherapy* —Cultures should be made at once The sensitivity of the organism to the various chemotherapeutic agents should determine which one is to be employed If indicated, gas gangrene antiserum should be administered in therapeutic doses

(2) *Incision and Drainage* —A local incision should be made to drain any collections and pockets must be opened

(3) The patient should be in *isolation* and should be given supportive therapy

(4) *Blood transfusions* are of great value They should be given regularly in small amounts, *i e*, 250 cc every two days The purpose of such blood transfusions is not to correct an anemia but to supply some vital defensive substances that are not present in the patient at that time As the host destroys the transfused blood in a few days, it is necessary to replace it constantly to maintain the defensive factors An adequate blood count in such infections does not mean that blood transfusions are not necessary

Where these so-called conservative measures have not been successful or where the infection continues to spread, radical amputation of the part may be indicated and may be lifesaving

While it is necessary to perform a major amputation in many patients with clinical gas gangrene infection, the mere presence of *Cl perfringens* or *welchii* in the tissues, particularly in elderly individuals, does not mean that clinical gas gangrene infection is present These organisms inhabit the colons of elderly persons As these patients are frequently bedridden for long periods of time and at times are incontinent or not too hygienic, the gas organisms may be driven into their skins and cultured from any open wound

The symptoms of gas gangrene infection should be kept in mind when the question of amputation is raised in cases of arterial obliteration by infection These are clinical symptoms and are not based merely upon the culture of the organism These symptoms are (1) fever, (2) increased pulse rate, (3) sanguinous drainage from the wound; (4) massive edema, (5) an anxious sick look and malaise greater than a local infection usually causes, (6) culture of gas organisms from the wound—when the culture is positive and the above symptoms occur, then clinical gas gangrene infection is present, (7) roentgen ray and clinical evidence of gas formation in the tissues

An example of the importance of this differentiation is my experience during World War II with a group of patients received aboard the U S Naval Hospital ship *Relief* for emergency amputation These patients had been wounded during the invasion of the Island of Peleliu Cultures made in frontline hospitals or on evacuation ships were positive for gas gangrene organisms

Although these patients had a positive culture of these organisms and gas of some type was present under the tissues, clinically they did not have a gas gangrene infection In some, the crepitation was caused by air forced into the tissue by a high-velocity missile The positive cultures were the result of contamination of the skin by the human excreta which the Japanese used for fertilization

Their treatment included incision and drainage of any pockets. The wounds were left open and sulfonamide power was applied locally. Enormous doses of penicillin (6 000 000 units per day) were given. To this were added prophylactic and therapeutic injections of gas gangrene antiserum and repeated small blood transfusions.

No major amputation was performed. All are walking today on legs that they would have lost had merely the evidence of the presence of gas gangrene organisms in the culture or air in the tissues been sufficient to dictate the amputation.

This example is not intended to devaluate the importance of a major amputation in the presence of an active gas gangrene infection. It is emphasized that gas gangrene infection should be diagnosed clinically in conjunction with the laboratory and not by a bacteriologic or *x*-ray examination alone.

(5) *Anticoagulants* should be used.

The invasiveness of hemolytic streptococci and the marked reaction of the tissue to them with early edema may cause an occlusion of a major artery with gangrene. The measures detailed should be instituted and the patient observed from twenty-four to forty-eight hours. If there is no evidence of localization and if the infection is spreading or uncontrollable no delay in amputation is to be countenanced.

## ACUTE OCCLUSIONS OF THE UPPER EXTREMITY (CERVICAL RIB, SCALENUS ANTICUS SYNDROME, OTHER OBSTRUCTIVE SYNDROMES)

Acute arterial occlusion in the upper extremity often is caused by the scalenus anticus syndrome with or without a cervical rib. This problem will be discussed fully on pages 470 to 480.

The scalenus anticus syndrome is seen most frequently at the time of puberty or rapid growth development. Patients turning from a sedentary life to one of muscular activity may acquire the lesion. Thus during war time there was an increase in the number of patients with hypertrophy of this muscle.

The scalenus anticus muscle as a result of hypertrophy or if distorted by a cervical rib or a fibrous band causes pressure on the subclavian artery and/or the brachial plexus. The symptoms may be acutely initiated by some sudden jerking motion in which the scalenus anticus muscle is put on tension. The subclavian artery, which passes between the scalenus anticus and the scalenus medius muscles, is then pinched in a scissor-like fashion so that its endothelium is injured and thrombosis results. There also may be pressure on the brachial plexus. Many of the symptoms therefore of an acute arterial occlusion in the upper extremity are accompanied by neurologic findings.

Treatment of these patients includes: (1) Division of the scalenus anticus muscle. This muscle is an anlage and is no longer necessary for our functions. It can be divided at its tendinous insertion on the first rib. Its division usually releases the subclavian artery and the brachial plexus from any untoward pressure.

(2) Where a cervical rib is present and is causing considerable pressure, it may be necessary to rongeur it away In this respect, it should be emphasized that any undue manipulation of the brachial plexus may set up a brachial neuritis with alarming palsies and attendant causalgia

(3) A fibrous band from the first rib or from the vertebrae to the first rib or an anomalous first rib may cause pressure in a similar fashion. In such cases, it is necessary to divide this fibrous or tendinous band In some, the scalenus medius muscle must be divided to liberate both the subclavian artery and the brachial plexus Some bone may have to be removed

(4) Other acute arterial occlusions may follow obstructions Thus a bony overgrowth or distortion following fracture of the first rib, clavicle or sternum (steering wheel injury) may compress a major artery, particularly the subclavian A tumor mass can have a similar effect Malposition such as occurs with hyperabduction or costoclavicular syndromes or with the abnormal position supplied by casts, or in the operating room, has caused vessel closure The treatment of such pressure requires its corrections primarily If thrombosis has occurred, arteriotomy and thrombectomy may be necessary. Often in the upper extremity, however, there is sufficient collateral circulation to take over the blood supply. This may be augmented by interruption of the sympathetics and the limitation of clot propagation by antithrombotic drugs Surgical or traumatic injury to the main blood supply of the upper extremity is given in injuries to arteries

Causalgia sometimes is a complication To relieve such pain, a thoracic sympathectomy of the type described in the chapter on Interruption of the Sympathetics, page 519, may have to be performed

## OCCLUSION DUE TO COLD

The occlusion due to frostbite, immersion foot, or trench foot has already been discussed on pages 270 to 282 In summary, the treatment consists of the following·

(1) The avoidance of all trauma to the part, with protection of any skin breaks and the use of sterile precautions in its handling

(2) The removal of all obstructions to the arterial or venous flow

(3) Definitive treatment for the cold exposure The exact method for treating cold injuries is under study in many clinics at this time It had been considered that the pathogenesis occurred from a sludging and slowing and finally thrombosis of the vessel with secondary tissue cell injury or destruction Experiments on animals appeared to indicate that the cold exposure injures the tissue cell primarily and that thrombosis of the involved vessels is secondary to such tissue cell injury If these conclusions are tenable in man, the injury from cold occurs on exposure In such event rapid warming of the part is the treatment of choice These animal experiments have not explained the occasional complete recovery, even at deep freeze temperatures, of a part or the hibernation phenomenon in animals Both methods of treatment are mentioned briefly.

(a) *Rapid Warming Method* —In such therapy, the part exposed to cold is brought back to normal room temperature in a period of a few hours The part is *never* heated. Supportive therapy, in general, as well as the

anticoagulant drugs are then used In the animal experiments there was no greater and probably less incidence of tissue destruction and gangrene when this method of therapy was employed

(b) *Slow Warming Method* —This method assumes that there is considerable damage to the blood supply to the part and that many tissue cells can be saved from injury even after the exposure The basis of this therapy is the avoidance of warming of the part until the success or failure of the recanalization or collateral circulation is assured In this method the *cooling* of the part is important and should be continued as long as it is necessary for the decision of surgical management to be made

The basis of this treatment has been discussed It is derived from the fact that when blood vessels have been occluded for a considerable time the venous and lymphatic return from the affected extremities is impaired and is inadequate to take over the increased arterial load which follows warming of the part

This therapy restricts the arterial supply to the part to that quantity which the venous and lymphatic drainage can carry away until such time as the two processes have become equalized This may take considerable time and therefore it may be necessary to cool the part for several weeks

When too much arterial blood enters the limb edema duskiness and blebs appear Later signs are blood filled blebs necrosis of the skin and gangrene

The affected part should be exposed in a cradle or tent with ice bags around it and electric fans used to circulate this cool air In this way a temperature of approximately 55° F can be maintained As the circulation of the part improves the number of ice bags may be reduced

(4) The anticoagulant drugs should be used if other injuries or wounds do not contradict them Anticoagulant therapy is of extreme importance in such patients as the work of Crossman[1] and Blalock[2] has clearly shown Their investigations proving that a much lower temperature is required or more trauma is necessary to produce gangrene in a refrigerated limb when anticoagulant treatment is given are significant

(5) Sympathetic blocks in patients seen shortly after exposure appear to reduce the incidence of necrosis Those seen days or weeks after such an exposure do not seem to be improved by such therapy There may be residual symptoms after the part has been saved Causalgia and hyperhidrosis may develop and require the performance of a sympathectomy to achieve relief Other rehabilitative measures such as physiotherapy modalities are of help

## MEDICAL MANAGEMENT OF ALL ACUTE ARTERIAL OCCLUSIONS

Acute arterial occlusion regardless of its etiology requires excellent medical supervision even though the primary treatment may be surgical

The principles of this treatment are

(1) **The Relief of Pain.**—This is of great importance not only because pain must be relieved to make the patient comfortable but because the pain in acute arterial occlusion is a cause of spasm Morphine which is

readily available, may be given in a dosage of ½ grain (0.803 gm.) at two to three hourly intervals. Demerol also may be used in dosages of 200 to 300 mg. Of the numerous other drugs available for the relief of pain, aspirin, which is both a good analgesic and a local vasodilator, papaverine and whiskey are of value.

(2) **Anticoagulants.**—Heparin should be begun at once as detailed in the chapter on Antithrombotic Therapy, page 651. In general, 50 mg. of heparin are injected intravenously at once, followed by an injection of 50 mg. into the deep subcutaneous tissues at the end of an hour. The dosage thereafter is 30 to 70 mg. every three hours injected into the deep subcutaneous tissues, depending entirely upon the coagulation time, which should be kept between twenty and forty minutes. In the acute stages, heparin may be given by intravenous injection. The average patient requires 30 to 50 mg. per hour to maintain a clotting time twice that of normal. The necessity of making accurate and continued laboratory tests complicates the procedure.

One of the oral anticoagulants, such as Dicumarol, Tromexan, or the others, should be started at once in dosages as described in the chapter on Antithrombotic Therapy, page 651. In general, 300 to 400 mg. of Dicumarol can be given at once, followed by 100 mg. daily thereafter. The subsequent dosage is determined by the prothrombin time, which should be kept between 2 and 3 times normal. The dose of Tromexan is approximately five times that of Dicumarol.

(3) **Protection of the Extremity.**—It is important to protect the extremity against trauma, pressure, or skin breaks. Skin breaks may be the source of an infection, which may interfere with the patient's recovery. Blebs, fungus infections, and other skin breaks should be surgically dressed. Care should be used in moving the part, because where the skin is partly devitalized, the hand may slide along the skin and cause a break. Blisters should be kept intact. No antiseptics should be applied to such extremities. Great care should be taken in cleaning them. It is best to wrap the part in a thick layer of cotton or candy cotton.

(4) **The Temperature of the Extremity.**—The question of the optimum temperature to be applied to an extremity with an acute arterial occlusion is still being debated at the present time. In general, we believe that an ordinary environmental temperature of 70° F. is optimal. The control of the temperature has been discussed previously.

Where the affected part seems to be lost, cooling has returned the circulation in a few cases. The therapy in such cases is similar to that of refrigerated limbs.

(5) **Position of the Extremity.**—The position in which the extremity is placed is determined by the degree of edema that is present and the color of the part.

If there is marked edema and some cyanosis, very slight elevation of the extremity may improve the circulation. In like manner, if the part is pale and cold and there are signs of atrophic changes, putting the limb in a position slightly below the cardiac level may improve the circulation by gravity. For this purpose, Saunder's oscillating bed with the varied positions it makes possible may be of value. If this bed is used, the ratio of raising and

lowering the bed should be regulated carefully by observation of the changes in the limb When the part is raised it should be left in this position only until the venous blood has drained out of the limb and then lowered When the part is lowered it should be kept down only until it has become suffused but not to the point of cyanosis or edema

(6) **Drugs** —The use of drugs other than the ones described, has not been too effective except for those drugs which are designed specifically for interruption of the sympathetics

(7) **Interrupting the Sympathetics** —The paravertebral sympathetic nerve block is an effective method and can be applied quickly by any trained physician Novocain is used 5 cc. of a 2 per cent solution being injected in the paravertebral area of the second third and fourth lumbar vertebrae for the lower extremity and in the stellate ganglion for the upper extremity A polythene tube can be introduced through the correctly placed needle and the needle withdrawn Procaine then can be injected from time to time without the reintroduction of the needles The area of injection must be kept sterile

The drugs such as tetraethylammonium chloride Dibenamine and Priscoline have been discussed Their actions and shortcomings are detailed on pages 491 and 492 in the chapter on Interruption of the Sympathetics

Intravenous ether and novocain will produce a sympathectomy effect which is described on page 490 of the same chapter The effect of these drugs is more limited than is the paravertebral nerve block

Spinal anesthesia in the absence of other sympathetic interruption procedures will help for a short time

(8) **Mechanical Apparatus.**—Suction pressure boots intermittent venous occlusions and other modalities of this type have been discontinued in most clinics because they are of questionable value and have a traumatizing effect The whirlpool tank has been used with beneficial results especially to atraumatically cleanse the part

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Chapter

# 24

# ARTERIAL AND VENOUS OCCLUSION OF THE MESENTERIC VESSELS

MESENTERIC thrombosis, fortunately, is a rare but deadly cause for intestinal obstruction. The classical descriptions of the lesion were derived from postmortem examinations, since the early patients uniformly died of this intra-abdominal catastrophe.[5,21] Reasons for these gloomy results were twofold. The diagnosis was rarely made preoperatively (4 per cent)[21] and the pathology was so extensive that resection of the involved bowel carried a high mortality. A direct attack on the thrombosis rarely has been attempted. By mesenteric thrombosis is meant the occlusion of the mesenteric vessels, the artery or the vein or both, with the loss of the vascular supply to the part of the bowel supplied by these vessels. This is followed by avascularity, a closed loop intestinal obstruction, and a spread of the obstruction to other loops of bowel.

**Etiology.**—Mesenteric thrombosis results from occlusion of the mesenteric arteries and/or veins. Up to 1948, of the 616 cases collected by McClenahan and Fisher,[18] 43, or 7 per cent, had been treated successfully. The age incidence is from twenty to seventy years with the average of forty-three years. Fortunately, this condition is an uncommon cause for intestinal obstruction, only 5 of 235 cases being due to mesenteric vessel occlusion (2 per cent).

1. **Arterial Obstruction.**—The superior mesenteric artery most often is involved in arterial obstructions because it comes off the aorta at a higher level than the other mesenteric vessels. Such mesenteric arterial obstructions are caused by the following:

(*a*) In the past, *embolism* has been the most common cause of occlusion of the mesenteric artery. Trotter[21] found that 128 of his series of 187 cases of mesenteric arterial occlusion were caused by embolism. In embolism, there is usually a left-sided heart disease of the rheumatic type with auricular fibrillation. The clot arises in the auricular appendage or in the mural walls of the heart. Its movement frequently follows a surgical operation, a coronary occlusion, an obstetrical delivery, or some other irritating cause. The dislodged embolus then occludes the mesenteric artery.

(*b*) *Direct or indirect trauma* to the abdomen if severe may initiate a local thrombosis and cause a mesenteric arterial occlusion.

(*c*) *Arteriosclerosis* or rarely *thromboangiitis obliterans* by plaque or calcification may narrow the mesenteric artery. The remaining lumen may be closed by sudden clotting.

(*d*) *Surgery* in which the mesenteric artery has been damaged or inadvertently ligated will cause such an occlusion

(*e*) *Extreme distention of the intestine* due to adhesions ileus tumor herniation intussusception or other forms of obstruction can increase the intra intestinal pressure sufficiently to cause venous or arterial vascular occlusion

2 **Venous Obstruction.**—Mesenteric venous thrombosis usually is a complication of acute intra-abdominal inflammation such as pelvic phlebitis, pelvic infection appendicitis diverticulitis and peritonitis [3] In the exhaustive collection of cases reported by Trotter[21] in 1913 60 per cent of the thrombosis occurred on the arterial side and 40 per cent on the venous side

The venous system is occluded secondary to such obstructions as *strangulation adhesive bands intussusception torsions and strangulated hernias*

In adynamic ileus the *distention* within the lumen of the bowel may be sufficient to cause a venous stasis and eventual obstruction Dragstedt[8] showed that when the pressure within a closed loop of the intestine reaches 60 mm of mercury the arterial circulation to that part of the bowel is blocked This occurs at even lower pressures on the venous side Noer and Derr[20] showed that intra-enteric pressures of 70 mm of mercury prevent filling of an intestinal vessel even if the vessel is intact Arteries begin to fill between 60 and 50 mm of Hg The capillary filling does not occur unless there is complete deflation (under 10 mm of Hg ) [20]

**Symptoms** —The symptoms of mesenteric thrombosis following an acute arterial occlusion usually develop suddenly are frequently shocking and intestinal obstruction follows in a short time If the block is small and a collateral circulation develops the symptoms may be mild and at times may disappear This is rare when the obstruction develops from occlusion on the arterial side

Symptoms of obstruction on the venous side may be mild for a considerable length of time It has been noted by Wangensteen [23] Babcock [1] and others that the condition may go undiagnosed for as long as a week

Whether the obstruction is on the arterial side or the venous side there are increasing signs of *distention constipation* and *cramp-like* pains usually not localized There is an increased *peristalsis* in some areas with other parts of the bowel silent *Vomiting* and at times *diarrhea* follow As a result of hemorrhage into the lumen material in the diarrhea may be blood tinged or of a wine color At times the vomiting becomes blood-stained

The *temperature* does not rise until there is a secondary *peritonitis* Peritonitis usually is a late sign and indicates beginning perforation The *intestinal pain* may be agonizing

*Shock* is a common symptom and is attributed by Wangensteen[23] to the blood loss into the bowel It may be explained on a reflex sympathetic basis

The *roentgen ray* picture is usually typical showing fluid waves and distention This distention is more prominent in the upper intestine if the superior mesenteric vessels are involved At a later stage there will be more distention a loss of haustration and fluid levels in many areas in the

abdomen The so-called "stepladder" appearance of the abdomen is an extremely late sign Anteroposterior, lateral and oblique x-ray views should be taken in both the upright and supine positions

**Pathology.**—The pathologic picture in the arterial mesenteric thrombosis is usually that of an embolus Secondary thrombosis occurs both on the arterial side and on the venous side This thrombosis is due to secondary congestion following the arterial block

The artery at the site of the embolus lodgment is in spasm, as are the collateral vessels Many of the symptoms and the progression of the disease may be on the basis of this spastic factor

The fixation of the embolus by *spasm* at the point of lodgment has been proven many times This was demonstrated in 1932 by Gosset,[10] in 1935 by Allen and MacLean,[1] and more recently by Laufman and Method [15] This spasm factor is of such great importance that it will be discussed later under therapy With the occlusion of the artery, the vein is also occluded After the occlusion occurs, there is a progression of the blood from the arterial side of the capillaries to the venous bed, until gradually the arterial side is empty and the venous side more congested [8] This is probably due to the spasm on the arterial side This spasm apparently continues until all hope of recovery from the arterial occlusion is past, at which time the spasm relaxes and some of the blood leaks back When the spasm is released, the same condition exists as that which follows the relaxing of blood vessel spasm in an extremity There is a so-called reactive hyperemia, which Lewis[16] attributes to the inflow of blood into an area which had been avascular In the arterial thrombosis due to arteriosclerosis there may be all degrees of calcification and plaque formation in the arteries Often a proximally propagating clot is present and this may involve the collateral radicals The changes in the bowel wall vary with the extent of the occlusion

If the spastic condition persists, infarction occurs, the degree becoming greater with the length of time the obstruction persists Eventually, there will be *edema, loss of color* with later *gangrene,* and eventually *perforation* if the condition is not relieved

In some cases after a small arterial embolism the collateral circulation takes over, and there is no infarction

When the occlusion is on the venous side, *symptoms of edema,* engorgement, cyanosis, and discoloration will be more severe than when the obstruction is of arterial origin This is due to the fact that the venous blockage causes at once a massive engorgement This blockage not only stops the venous drainage but also involves the lymphatic system This engorgement together with anoxemia causes a damage to the endothelial cells of both the blood vessels and the intestinal wall

After a relatively short time, the arterial end of the circulation will be affected secondarily, as the arterial blood is no longer able to pump against the increased resistance from the venous side in the capillary bed Thrombosis then occurs early on both the arterial and venous sides

**Prognosis.**—The prognosis varies directly with the length of time the obstruction is present The degree of bowel involvement varies with the

point of the occlusion and whether it is venous arterial or both One patient was seen in whom there was a venous occlusion with necrosis of all of the bowel from the ligament of Trites to the lower sigmoid This condition came on quite rapidly in a thirty two-year-old patient with extreme hypertension (245/160) and kidney disease

The symptoms were insidious in origin in a physician who had had only mild intestinal complaints for eight days and had continued his practice until two days prior to his admission and subsequent death The signs were so inconclusive that exploration was carried out against the dictates of some internists who argued that the symptoms were likely intestinal grippe At operation spotty necrosis of nearly 20 feet of bowel was found with thrombosis and diffuse peritonitis

**Diagnosis** —In an acute arterial mesenteric thrombosis the signs of pain distention and obstruction will be early and lead one to suspect the lesion If there is rheumatic heart disease auricular fibrillation and previous embolism with the abdominal symptoms the conclusion will be obvious The diagnosis of the insidious type of mesentric thrombosis frequently is difficult It is rarely made preoperatively and is discovered at exploration after considerable delay Most of the operations are performed on a diagnosis of possible surgical abdomen ' rather than that of mesenteric thrombosis

The soft dough like abdomen with tenderness which is persistent a leukocytosis intermittent periods of vomiting and some diarrhea the absence of fever and the absence of other causes for the complaint should make one suspect the pathology

The degree and extent of the thrombotic process may not be apparent early If the operation were done too early a loop of bowel which might recover may be sacrificed or a section of bowel which could become gangrenous later might be left

At *operation time* the diagnosis is made when the abdomen is opened *There is bloody fluid free in the peritoneal cavity The bowel may be all* shades from a duskiness to black but usually is a cyanotic bluish-gray and nonglistening In the portions in which obstruction is present peristalsis is inactive Pulsation on the arterial side may or may not be present

The importance of the bowel temperature as a prognostic and therapeutic aid has been stressed before and will be discussed under Treatment.

**Treatment.**—(*a*) SURGICAL RESECTION —Treatment for *mesenteric* thrombosis is surgical once the diagnosis is made or suspected with excision of the involved area of bowel and its mesentery The first reported enterectomy was by Elliot in 1895 [8]

In advanced stages the safest resection in these patients is by exteriorization This is important particularly if there remains a question as to the extent of the disease Where the occlusive process is of a spotty type exteriorization of more bowel is indicated

The value of surface temperature of the bowel as a criterion of its possible recovery has been emphasized In questionable cases this method may be of value Experimentally after two and a half hours of obstruction the bowel temperature drops 6 degrees If after release of the strangulation

the temperature begins to rise, the bowel will recover. This measure may determine the necessity or degree of resection necessary.

In early cases where the demarcation is well delineated, resection and primary anastomosis is the treatment of choice. Anastomosis is made usually in an end to end fashion with one or two layers of silk for the serosa. The cut mesentery should be approximated to prevent obstruction.

(*b*) OTHER TREATMENT.—1. *Sympathetic Interruption.*—It is apparent that the pattern of response following main-stem vessel occlusion is due to spasm.[7,17,23] Sympathetic interruption thus is indicated on a regional basis. This can be done with blocks or sympathectomy. Injections of novocain in the mesentery as suggested by Herrlin[11] may help.

2. *Anticoagulants.*—Sludge formation occurs early after obstruction.[17] It can be prevented by anticoagulants. These drugs should be used routinely. See Chapter on Antithrombotic Substances.

3. *Drugs.*—(*a*) *Papaverine Hydrochloride.*—This drug can release some of the arterial spasm and venous occlusions if used before thrombosis occurs.[17]

(*b*) *Sympatholytic Drugs.*—Tetraethylammonium chloride requires shock-producing doses to equal sympathetic blocks. It may help some of the venous occlusions but is not consistent. Priscoline and Dibenamine are in the same category. Oxygen therapy has not shown itself to be of value.[3]

The old tests for bowel viability, namely the return of peristalsis, the normal color of the bowel, and the return of pulsations to the part, are still primary criteria and should not be abandoned.

The tests of Lange and Boyd[13] with fluorescein, while accurate in most cases, are impracticable for operating room use. The poor results after resection of bowel for mesenteric thrombosis probably are caused by the difficulty of determining the point of demarcation.[4,5,11] While the mortality has dropped from 94 per cent in 1944 to 93 per cent in all the cases up to 1948, the mortality in the definitive operative treatment remains 70 per cent.[18] In those patients who recover after massive resection, so little of the small bowel remains that they may be crippled by diarrhea. The happy result of Meyer's case is the exception.[19] Klass[12] performed an embolectomy for mesenteric occlusion. His patient died of other causes but apparently with a viable bowel. In case of a sudden abdominal pain with shock and a bloody stool, one should consider immediate laparotomy, especially if the patient is a fibrillator. More recently there has been an attempt at direct intervention with removal of the thrombus at its site. It is not unlikely that with better diagnosis this method can be extended and may be practicable in a certain percentage of patients. The time limit will have an important bearing on the application of this surgical procedure.

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Chapter

# 25

# NEUROVASCULAR LESIONS OF THE UPPER EXTREMITY

*Cervical Rib; Scalenus Anticus Syndrome; Costoclavicular Syndrome; Hyperabduction Syndrome; Malposition Syndrome*

THE subclavian artery arises from the innominate artery on the right side and from the aorta on the left It courses through the neck for a distance of approximately 6 centimeters before dipping into the comparatively narrow space between the clavicle and the first rib to enter the axilla and become the axillary artery

This subclavian artery, in its usual course, runs between the scalenus anticus and the scalenus medius muscles The scalenus anticus muscle thus divides the subclavian artery into three parts [10]

The subclavian artery may pass through the substance of the scalenus anticus muscle The cords of the brachial plexus pass through this same muscular and narrow bony area, but are placed more posteriorly and laterally

In abnormal positions of the arms, shoulders or neck, these structures may be subject to pressure The frequent readjustment of the head, arm, and shoulder posture while asleep in bed is an example of reaction to such pressure

If work or posture makes the pressure continuous, then well-defined syndromes develop

With this brief review of the anatomy of the area involved in neurovascular lesions of the upper extremity, certain of these syndromes will be described

## CERVICAL RIB

While cervical rib was known to the early physicians Vesalius and Galen,[24] the first modern description of the cervical rib syndrome was made by Willshire in the *Lancet* in 1860 [24a]

The reason for the development of symptoms from the cervical ribs is not entirely clear, but theoretically may be explained in two ways According to Todd,[22] the symptoms arise because of an abnormal shoulder girdle development If there is a greater descent of the shoulder girdle or an arrested descent of the sternum, the subclavian vessels and the brachial plexus may be compressed over an abnormal cervical or at times a

first dorsal rib. Todd explained the more frequent occurrence of cervical rib in the female by the failure of development of suspensory shoulder muscles, especially the trapezius, and also by a poorly developed rectus abdominal muscle which does not pull the sternum caudally.

Jones[12] attributed the neurologic symptoms of cervical rib to an abnormal development of the brachial plexus. He believed that if the brachial plexus originates mainly from the lower cervical or thoracic vertebrae the nerves will be angulated and compressed over the cervical or first dorsal rib and then will produce symptoms. This theory explains the nerve but not the arterial symptoms.

**Abnormal Bone Ossification Theory (Pratt)** [21]—The anatomy of the bones involved in pressure on the artery or nerve has not been stressed. The clavicle ossifies earlier than any other bone in the body. This growth occurs from three germinal centers. The two primary ones on the medial and lateral area begin the fifth week of fetal life, while the secondary center for the sternal end does not appear until the eighteenth year and does not unite with the rest of the bone until the twenty fifth year. Any abnormality in these developments could intrude on the already limited bone space. The *years of final growth* of this bone correspond with the greatest incidence of the lesions.

The ribs have four ossification centers. The center for developing the epiphyses for the head and tubercle appears between the ages of 16 and 20 and unites with the body about the twenty fifth year. An abnormality in growth may be a factor. In addition those who perform arduous tasks develop thicker more curved clavicles with larger notches for tendon attachments according to Mall[14] and Fawcet[8].

The author was impressed with the large size of the first rib even in women developing the syndrome and has called attention to its presence. This bone encroachment by its physical size in an area already small for the important structures passing through it may well be a factor in the pressure syndromes. Thus any abnormal overgrowth or delayed development in the formation of the clavicle the cervical or first rib could result in a disproportion of the relative size of the space between the rib and clavicle and produce pressure.

**Etiology**.—The incidence of cervical rib is rare (31 in 80 000— 038 per cent routine autopsies)[2]. At the Mayo Clinic from 1910 to 1926 there were 303 cases of cervical rib syndrome in 540 413 admissions an incidence of 054 per cent. Two of every three patients were females. Of a total of 303 cervical rib cases 219 or 72 per cent were females. 25 out of 36 cervical rib operations were performed on women. When Halstead[11] reviewed the literature in 1916 he found 716 recorded cases of cervical rib syndrome.

Between 65 and 80 per cent of these patients have no symptoms. The signs develop most often at adolescence or middle age which is the time of hypertrophy and atrophy of muscles. The onset of symptoms frequently is associated with trauma or an overuse of the neck and shoulder muscles. The condition is seen particularly in muscular individuals.

The symptoms are caused by pressure of the scalenus anticus or scalenus medius muscles or by a forward protrusion of a cervical rib or fibrous band. Hypertrophy of the muscle itself may constrict the subclavian

artery and/or the brachial plexus by a scissor-like action At times, the subclavian vein, which normally passes in front of the scalenus anticus muscle, may be traumatized against the clavicle by similar action of the scalenus anticus muscle This will be discussed later

The part that trauma may play in the development of this syndrome is apparent Severe exertion, particularly of the neck, head, or upper parts of the chest, and shoulders, can cause an acute onset of the symptoms by this sudden pressure

In war times, many persons change from light to heavy work The high wages and patriotic necessity bring individuals into the shipyards and munition plants or onto farms where they use muscles previously not developed This is true particularly in the female workers The number of patients with scalenus syndromes likewise increases. We have seen acute subclavian arterial thrombosis following heavy calisthenics, after the severe strain of chopping a heavy tree, and following the use of a pressure hammer for a long period of time. It has developed after the effort of a peddler to force his pushcart over a very high curb It has followed loading ammunition during a barrage In one patient who recently became a farmer, symptoms came on acutely after pitching hay all day In most instances, however, there were prodromal symptoms, either of arterial supply failure or nerve pressure, before the exciting cause—the trauma or exercise—occurred

The symptoms of cervical rib and scalenus anticus syndrome may be similar

**Anatomy.**—To understand the symptomatology of the cervical rib syndrome, it is necessary to review the anatomy of this area The scalenus anticus muscle arises from four tendinous slips from the anterior tubercles of the transverse processes of the third to sixth cervical vertebrae It inserts as a flat tendon into the scalene tubercle near the inner border and on the upper surface of the first rib, anterior to the subclavian groove of this rib

The scalenus medius muscle arises in six slips from the posterior tubercles of the transverse processes of the lower six cervical vertebrae It has a wide insertion into the upper surface of the first rib between the scalene tubercle and the subclavian groove

The scalenus posticus muscle originates from the posterior tubercle of the lower two or three vertebrae and inserts on the outer surface of the second rib It is usually too far posterior and lateral to be involved in pressure on the brachial plexus or the major arteries This scalenus posticus occasionally may be fused with the medius

These normal origins and insertions of the scalenus anticus and medius muscles may vary, a slip from the scalenus anticus passing posteriorly to the subclavian artery being one of the most common anomalies This muscular band may cause definite arterial deficiency symptoms

These muscles are supplied by branches from the second to the seventh cervical nerves Their muscular action is two-fold. When action is initiated from above, they act as accessory inspiratory muscles by raising the first and second ribs When initiated from below, these muscles help to bend the vertebral column laterally When acting jointly, they aid in slightly flexing

the spinal column The subclavian artery normally passes between the scalenus anticus and the scalenus medius laterally The brachial plexus also descends between these two muscles but more laterally

While these scalene muscles were essential when men were quadrupeds and were dependent upon their heads and necks for moving objects they no longer are essential

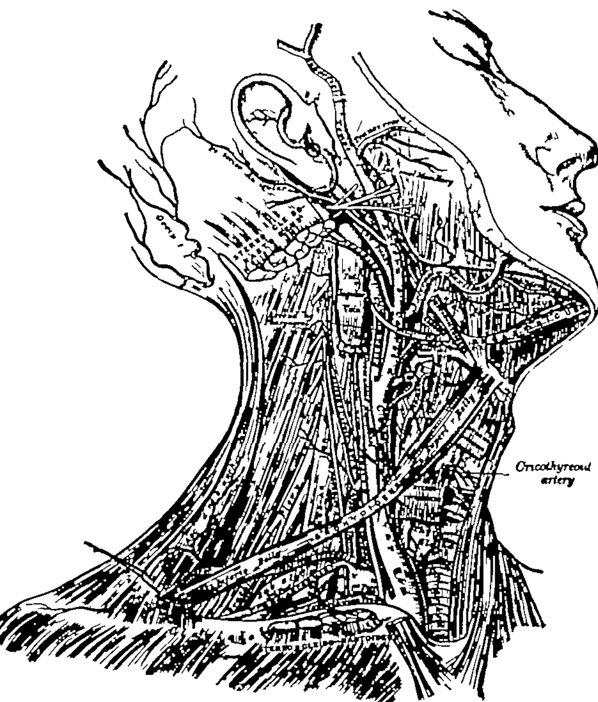



FIG 147 —Anatomy of the brachial plexus and subclavian artery and vein. (Gray's Anatomy ) Pressure on these structures causes vascular neurological or combined types of syndromes.

**Symptoms.**—The symptoms of the cervical rib syndrome may be vascular or neurologic or both, depending upon the amount of pressure and where it is exerted The signs may be temporary, intermittent or continuous

1 *Circulatory Symptoms* —The circulatory symptoms vary with the degree of occlusion, the amount of spasm, and the length of time the lesion persists. The signs may be mild for a long time

There is frequently a *numbness* and increasing *weakness* in the hand, with often a *blanching* and *coldness* This is evident particularly with weather changes. With acute or continuing obstruction, the circulation may be entirely cut off with thrombosis The blanching may be followed by *mottling* and at times even by *gangrene.*

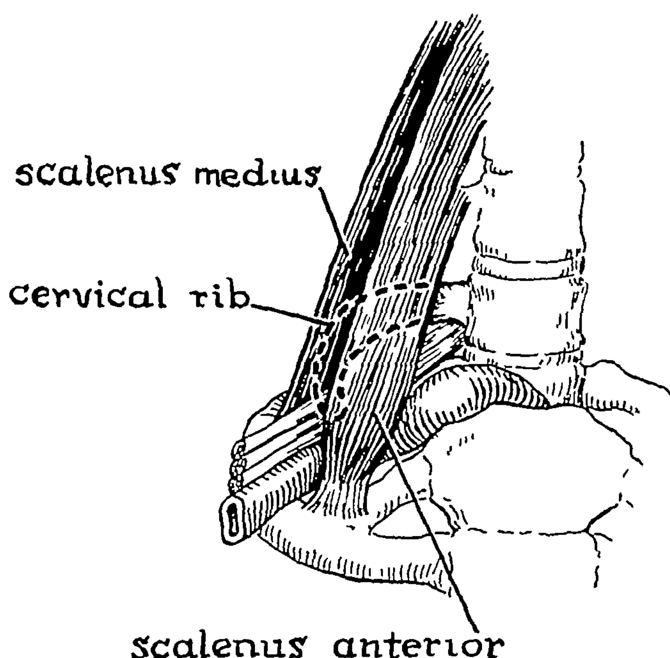



FIG 148 —Anatomy of scalenus anticus syndrome with and without cervical rib The vital nerve and arterial structures pass between the scalenus anticus and medius Hypertrophy of the muscle, displacement of the muscle by a rib or other rudimentary structure, over-growth of bone or tumor growth may cause pressure on one or both of these parts with resultant symptoms

Spasm at once is a factor If spasm is controlled and adequate collateral circulation develops, the symptoms may subside if the cause is eliminated In complete occlusion, the pulse is absent and the blood pressure and oscillometric reading zero Once this occurs, the original circulation rarely is re-established.

2 *Neurologic Symptoms* —*Numbness* and *tingling* begin in the finger tips, hands and arms *Pain* is a consistent sign. These symptoms are more severe during activity *Electric-like shocks* may occur In the chronic forms, *muscular atrophy* and even *paralysis* may develop, although usually the circulatory changes have made the condition acute before this advanced neurologic change is complete The symptoms are aggravated by exercise or motion placing the scalene muscles on tension

The most common symptoms are *weakness* of the part *pain paresthesia numbness stinging pain* and *coldness* When the cervical rib is on the right side there are more apt to be symptoms as Murphy[16] pointed out This is due to the fact that there are more right- than left handed people and when one is right handed the right shoulder tends to drop Anatomically too the right brachial plexus is closer to the ribs than on the left side and thus more subject to pressure

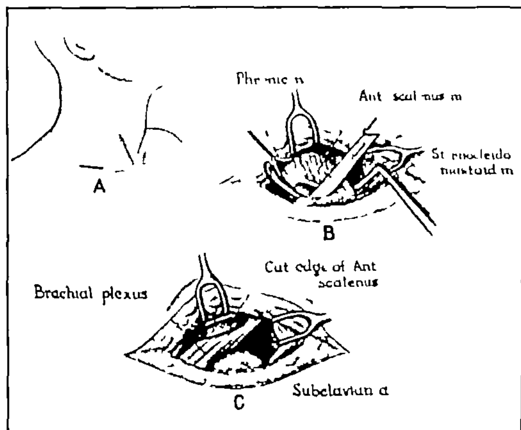



FIG 149 —A Site of incision for scalenotomy This transverse incision above the clavicle begins just over the lateral edge of *the clavicular insertion* of the sternocleido mastoid muscle, which is retracted and not divided

B The scalenus anticus muscle is isolated. A silk suture retracts the phrenic nerve and the scalenus anticus muscle is divided at its rib insertion The muscle has been elevated on a dull ligature carrier which delivers it into the wound for safer division on tension.

C Cut edge of scalenus anticus muscle. Note that the brachial plexus and the subclavian artery are now in the field Where there has been pressure, the site of pressure can be visualized.

Embryologically accessory ribs also develop in the lumbar or cervical regions A so-called bicipital rib resulting from the fusion of the cervical rib and the first thoracic rib may cause symptoms More than one cervical rib on the same side of the neck have been noted

Patients have been seen in which the only symptom was pain or anesthesia in the course of one small nerve such as the deltoid or scapular nerve

**Diagnosis.**—The diagnosis of cervical rib is made from the physical signs and by exclusion of occlusive and primary spastic lesions At times the rib is protuberant or palpable The diagnosis of cervical rib is rarely difficult if the condition is kept in mind Roentgen ray findings of the rib or of an enlarged transverse process, with the symptom complex, complete the evidence

A small or poorly calcified rib may cause even more symptoms than a large one This is due to pressure on the plexus by a sharp point A fibrous or bony connection to the first rib or to the clavicle or a vertebra also may cause the syndrome

The diagnosis at times must be made by exclusion In case of x-ray doubt, where symptoms persist, surgical exploration for diagnosis is indicated Such exploration should not be delayed until permanent nerve or artery changes have occurred

**Treatment.**—If the symptoms are absent or slight, no surgical treatment is necessary In mild cases, corrective exercises, change of employment and postural exercises may be sufficient to relieve the symptoms If this therapy is not effective, or where symptoms continue or progress, surgical intervention is indicated

Lister[14] is reported to have removed a cervical rib which prevented an Army major from firing his rifle Coote[6] removed a cervical rib in 1861 Keen[13] reported that there had been 42 cases of cervical rib operated on up to 1906

It remained for Adson's[1] work in 1927 to bring to attention the importance of the scalenus anticus muscle as a pathologic and a therapeutic factor He showed that scalenotomy alone cured many of these patients Further contributions to our knowledge of the scalene muscle spasm factor were made by Ochsner, Gage, and DeBakey [19]

## SCALENOTOMY TECHNIC

The anterior approach to this area is advocated whether the scalenus muscle only is to be divided or the rib also is to be removed

*Anesthesia*—A local or an intratracheal anesthesia is used Local anesthesia is a satisfactory agent for this operation if general anesthesia is contraindicated This permits the cooperation of the patient in turning the head or shoulder to better deliver the muscle or other involved structures

If it is used, the anesthesia is introduced through a single wheal raised just above the clavicle and over the lateral edge of the clavicular insertion of the sternocleidomastoid muscle The incision site is anesthetized and the area blocked by infiltrating a triangular zone in the posterior triangle of the neck No further anesthesia is required

Since there is danger of injuring the pleura, an intratracheal anesthesia is safer This prevents pulmonary collapse and tension pneumothorax if the pleura is opened inadvertently

*Operative Technic*—The operation performed is a scalenotomy with or without resection of the offending rib A 5-centimeter incision is made transversely, just lateral to the clavicular insertion of the sternocleido-

mastoid muscle in the posterior triangle of the neck with division of the platysma myoides muscle The clavicular attachment of the sternocleidomastoid muscle need *not* be divided but is retracted medially The omohyoid muscle is retracted superiorly

*An avascular fatty space is dissected and the scalenus anticus muscle* exposed Branches of the transverse cervical and transverse scapular vessels are avoided These branches need not be divided and often are not seen So avascular is this space that it is not unusual to expose the scalene muscles without dividing a single vessel

The brachial plexus is lateral and should not be traumatized The phrenic nerve crosses the scalenus anticus muscle from the lateral to the medial side and serves as an anatomical guide It is more medially placed on the left side This nerve may be retracted and the scalenus anticus *muscle divided approximately at its tendinous attachment*

A dull ligature carrier inserted under the scalenus anticus muscle lifts the muscle into the wound The muscle then can be divided with a sharp scalpel a few fibers at a time safely on tension against the ligature carrier If the scalenus medius muscle is causing pressure it must be divided As the muscle is cut it retracts If a small vessel is encountered in the muscle it need not be ligated as the muscle contraction controls it There has been no annoying bleeding in any of the last 60 scalenotomies we have performed Adson s[3] use of a cautery at this point has not been found necessary Should there be bleeding in the muscle a brain clip will control it

Exploration for fibrous bands is made If the rudimentary rib itself is causing pressure it is removed with a rongeur Movement retraction or dissection of the branches of the brachial plexus should be avoided because a severe neuritis palsy or causalgia may follow any manipulation of the nerves in this area

**Sympathectomy With Scalenotomy** —Where the re-establishment of the circulation is questionable or where spasm is a factor a sympathectomy should be performed at the same time as the scalenotomy An endotracheal anesthesia is required as the pleura may be injured Sympathectomy can be done through the same anterior approach described for the scalenotomy operation but the results are not as consistently good as in other approaches The anatomy accounts for this variation

*Anatomy* —The inferior cervical ganglia lies between the base of the transverse process of the seventh cervical vertebrae and the neck of the first rib It is medial to the costal cervical artery These ganglia frequently coalesce with the thoracic ones The thoracic ganglia rest on the head of the ribs and are covered by the costal pleura White and gray communicans connect these ganglia with the spinal nerves The branches of the upper thoracic ganglia are small The third fourth and fifth are thought to carry cardiac accelerator fibers Branches of the second third and fourth ganglia enter the posterior pulmonary plexus[10]

*Technic* —After the scalene muscle is divided the pleura is stripped off the vertebrae with the fingers The sympathetic chain is dissected free By pulling the sympathetic cord up with a nerve hook it can be divided below the third thoracic level Division of the fibers of the chain connecting with the spinal cord and the second and third intercostal nerves is made

A similar procedure is followed with the dissection of the sympathetic cord and chain to the first thoracic ganglion (stellate) A section of the second and third intercostal nerves is removed to assure sympathetic denervation of the part (See page 518 )

The danger of some pleural injury and a pneumothorax must be stressed If this is suspected, it is better to open the pleura, leave a catheter in the pleura until the wound is closed, aspirate all air and remove the catheter while the anesthesiologist actively expands the lung This prevents tension pneumothorax.



FIG 150 —*X-ray picture of cervical rib with severe pressure symptoms*

## SCALENUS ANTICUS SYNDROME

Scalenus anticus pressure was noted by Murphy[16] in 1915 while resecting a cervical rib Adson[1] found that scalenus anticus resection relieved the symptoms of cervical rib without the rib's removal This suggested to Naffziger that the syndrome could be present without the rib, and he presented it as the Naffziger Syndrome [17]

A complete report of scalenus anticus syndrome with cervical rib symptoms in the absence of cervical rib was presented in 1934 by Ochsner, Gage, and DeBakey [19] In 1938, Naffziger and Grant[18] reviewed the literature and recorded 51 such cases The detailed report of Adson and Coffee[1] followed

**Etiology** —Hypertrophy of the scalenus anticus muscle is present in most of the cases An analysis of my patients with this syndrome showed that two-thirds of them gave a history of some trauma, unusual exercise or activity as an exciting cause

The part that spasm of the scalenus anticus muscle plays in the syndrome has been emphasized by Gage and Parnell [9] These investigators found that in 33 3 per cent of the patients the brachial nerves pass through the substance of the scalenus anticus muscle (20 per cent of the author's series)

That hypertrophy of the muscle plays a part is shown by the frequency of the condition in athletically developed individuals such as professional wrestlers weight lifters, and those who change from a sedentary life to one of muscular activity as previously described

**Symptoms** —The symptoms of scalenus anticus syndrome are those described under cervical rib (see page 474) In these patients often the symptoms may be relieved by relaxing the muscle

The oscillometric readings the pulse volume and the blood pressure are decreased by placing the scalene muscle under tension This is done by raising the arm and turning the head in the opposite direction This occurs in 15 per cent of normal individuals

If the symptoms are relieved by the injection of novocain into the scalenus anticus muscle the pathology is not cervical rib Relief after such an injection would appear to argue for the spasm of the muscle rather than its hypertrophy as the cause of the syndrome Muscle hypertrophy rather than spasm however is a much more common cause of the syndrome

When neurologic signs are present they are most often on the little finger side of the hand along the course of the ulnar nerve The ulnar nerve arises from the eighth cervical and the first thoracic nerves and therefore is more often involved

**Diagnosis** —The diagnosis of scalenus anticus syndrome is made on the symptoms Adson placed great importance on the test described by him in 1927

*Adson Test* —The patient sits upright with his arms resting on his knees He is asked to take a long breath elevate his chin and turn to the affected side A decrease or obliteration of the pulse or blood pressure is a positive finding

The diagnosis is made frequently by elimination of other causes for the symptoms. The condition may have been diagnosed as arthritis neuritis neurosis or pressure from a disc etc If the patient s work requires him to increase the tension of the scalene muscle on these structures the symptoms at such times may be diagnostic For example painting a ceiling placing items high above the head pitching hay or grain above the head or straining the shoulder girdle as in wrestling weight lifting car parking etc are all possible causes

Symptoms of arterial impairment or of nerve pressure without other cause are suggestive and warrant an exploration of the scalene area if arthritis neuritis or pressure from an aneurysm intervertebral disc or other pressure have been eliminated If Adson s test is positive exploration should be performed routinely

**Treatment.**—Scalenotomy relieves the symptoms. See pages 476 and 477 for a description of the technic The muscle and its fascia must be divided in its entirety for if a small segment is left it will increase the symptoms

Pressure in this area may occur from other causes besides muscle hypertrophy or bony growth There may be tumors in the neck or in the muscles etc. An identical picture simulating the syndrome developed in one of our patients due to a lipoma in the scalene muscle

Invasion of the scalenus muscle by other tumors may occur Other enlargements of the scalenus anticus muscle may produce similar symptoms

Taylor *et al* [21a] reported on a scalenus anticus syndrome caused by the invasion of that muscle by trichinosis The pathogenic Trichinella nematode in its encysted stage may invade any skeletal muscle On exposure of the muscle it was found to be studded with the parasites

## COSTOCLAVICULAR PRESSURE SYNDROME

The first description of the costoclavicular syndrome was made by Falconer and Weddell [7] Costoclavicular pressure is caused by the shoulders being pressed forward and downward and by hyperextension of the neck This syndrome was seen particularly during World War II when soldiers had to carry heavy packs The slave labor of the past where tremendous straining was required, such as in lifting heavy objects (pyramid stones) or galley rowing, produced similar syndromes Those affected at times dropped their burdens despite the known penalty for such an act These patients often are classified erroneously as having scalenus anticus syndromes

Since such vital structures as the main artery to the arm and the nerves of the brachial plexus pass through the narrow space under the clavicle and over the first rib, any malformation of the bony structures or malposition may cause pressure on the nerves or arteries in this area The syndrome should be kept in mind in the differential diagnosis of arm pain or atrophy.

**Symptoms.**—The symptoms of costoclavicular syndrome, like cervical rib, are circulatory or neurologic, depending on which structures are the site of the most pressure The symptoms are much like those described under cervical rib (See page 474 ) Usually, there are both circulatory and neurologic signs

**Diagnosis.**—The diagnosis of costoclavicular pressure is usually made by exclusion of similar syndromes The relief of symptoms when the malformation is eliminated, the absence of organic occlusion or disease, and roentgen ray evidence of the pressure in various positions are important diagnostic points

Practical criteria for the diagnosis of the costoclavicular syndrome are:

(*a*) Symptoms of pressure on the nerves and blood vessels of the shoulder

(*b*) Obliteration of the pulse and increase in the neurologic symptoms when the shoulders are dropped down and forward

(*c*) Relief of these symptoms with correct posture

(*d*) Indication that the patient assumes or maintains this faulty posture frequently

**Treatment.**—The treatment for mild cases of costoclavicular pressure consists of corrective exercises and postural changes

At times, sleeping postures must be varied so that undue pressure is not permitted on these structures If a patient's occupation causes the syndrome, it must be changed For example, if the patient paints ceilings, where there is a tremendous strain on the shoulder and extreme hyperextension of the neck, he must change his vocation If the condition continues to be disabling, a section of the rib or resection of the entire rib or clavicle is corrective

## HYPERABDUCTION SYNDROME (WRIGHT)

The hyperabduction syndrome has been described by Wright[25] as due to pressure following a phase of circumduction which brings the arms together above the head as a result of external rotation with the elbows flexed. Similar symptoms were depicted by Todd[illegible, 23] in experiments on himself over an eight year period which resulted in symptoms much like the syndrome discussed by Wright.

**Etiology.**—This pressure syndrome apparently is a result of an immoderate abduction of the arm in which some degree of rotation also occurs. This results in a neurovascular syndrome. Posture changes, unusual muscle exercises and habits, and certain types of work such as painting, automobile and airplane repair and other construction work which requires peculiar positions are important etiologic factors. Anatomical abnormalities in bone or muscle development may predispose to the development of the hyperabduction syndrome.

**Symptoms.**—Symptoms of this syndrome like others are both neurologic and vascular.

1. *Neurologic Symptoms.*—There is numbness and tingling beginning at the tip of the extremity and progressing proximally, particularly on the little finger side of the hand. There may be loss of muscle power.

2. *Vascular Symptoms.*—Vascular symptoms are those of pressure on the major vessels and prolonged ischemia. The symptoms vary from coldness and numbness with some degree of blanching to gangrene. These symptoms vary with the degree and the continuity of the hyperabduction. The relationship of this syndrome to Raynaud's phenomena has been emphasized.[8]

**Diagnosis.**—The hyperabduction syndrome must be differentiated from (1) scalenus anticus syndrome, (2) cervical rib syndrome, and (3) costoclavicular syndrome. This lesion must also be differentiated from (4) causalgia, (5) Raynaud's syndrome, and (6) organic arterial occlusion from pressure due to tumor or obliterative arterial disease.

*Test for Hyperabduction Syndrome.* The original test was made by moving the patient's arms through a 180 degree semicircle and determining whether the pulse diminished or was lost during the maneuver. Due to technical difficulty this was changed to record such changes with an oscillometer on the wrist. The pressure of the oscillometer was placed at the maximum oscillation and the readings are noted at 0, 45, 90, 135 and 180 degrees. Variations in the reading are therefore registerable.

The diagnosis of this syndrome is based upon (1) those with signs of the hyperabduction syndrome. These are in order of frequency: pain in the hand, numbness, paresthesia, Raynaud's phenomena, shoulder pain and weakness. (2) Hyperabduction causes obliteration of the pulse and also neurologic signs. (3) Hyperabduction position is assumed by the patient frequently. (4) Relief of the symptoms by release from hyperabduction.[8]

In testing for *scalenus anticus syndrome* the arm should be in adduction as this places the scalenus muscle on tension. Abduction tends to relax the scalene muscles and relieves the symptoms. This may be used as a differential diagnostic point.

Roentgen rays will demonstrate whether the patient has a *cervical rib* The other lesions can be differentiated by elimination

**Treatment.**—*Prophylactic treatment* is important in preventing the hyperabduction syndrome This is imperative particularly in children The position of the patient while working should be corrected In many cases, relief can be obtained by a change in posture and by care to prevent the hyperabduction position, particularly when sleeping Exercises may help by developing the elevator muscles of the shoulders to reduce the angle at which the structures pass between the first rib and scalenus anticus muscle

Operative correction may be necessary in some patients. Excision of a portion of the first rib or clavicle will increase the size of the area through which the neurovascular elements pass from the neck to the axilla

## MALPOSITION SYNDROME[21]

The author has called attention to the neurovascular lesions following bad or poor positions in the operating room The high incidence of neurovascular complications of abnormal positions on the operating table or in casts or extension apparatuses has brought this problem to the surgeon's attention.

The twisting of the arm above the patient's head, with constant pressure on the subclavian vessels and the brachial plexus, is one such example Anesthesia removes the warning pain symptoms While restraint is necessary, it should be accomplished by other methods than placing the hands under the patient, twisting them with restraining sheets or applying cuffs which are too tight

The position of the patient during a gynecologic operation or a rectal operation, with the limbs in the lithotomy position, also may cause considerable twisting of the vessels in the pelvis and the groin

In addition, the pressure of the leg holders or of the stirrups directly on the main vessel may cause additional damage to vascular and neurologic elements Those lesions following tourniquet applications have been discussed (See page 325 )

Treatment is prophylactic The position of the patient must be the surgeon's and anesthesiologist's responsibility and they should watch for these abnormal positions and the dangers involved Recognition of these dangers and their prevention have increased with the development of anesthesiology as a separate specialty

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Chapter
# 26
## VASCULAR DERMATOLOGIC LESIONS
### *Erythermalgia (Erythromelalgia)*

ERYTHERMALGIA is a condition in which the extremities become *hot, red* and *painful* due to vasodilatation. Such changes are seen also secondary to other diseases. Thus erythermalgia may be subdivided into *primary erythermalgia* unassociated with any other condition and *secondary erythermalgia* which occurs with many vascular diseases such as polycythemia, arteriosclerosis, or diabetes mellitus. The vasodilatation in erythermalgia was described as early as 1872 by Mitchell[2] and others. The most specific modern summary was presented by Smith and Allen[3] in 1938.

**Etiology.**—1. *Primary Erythermalgia.*—The cause for primary erythermalgia is unknown. The patient's main complaint is a burning pain in the palms or in the soles of his feet. Those affected most often are in the forty- to fifty-year-age group. The pain always is aggravated by exposure to heat. Many patients sleep with their feet uncovered. Others obtain relief by taking ice cold foot or arm baths.

2. *Secondary Erythermalgia.*—Erythermalgia may occur secondary to arteriosclerosis, hypertension, frostbite, polycythemia, heavy metal poisoning, gout, and carcinoma.

**Symptoms.**—In erythermalgia, the skin becomes a bluish-red color and develops a dark hue which is increased on dependency. The affected extremities are extremely warm and the circulation in the part, as shown by the major blood vessels, is greater than usual.

While the redness and cyanosis increase on dependency, they are present even on elevation. The temperature of the affected part may be registered with a potentiometer from 5 to 15 degrees above normal. There are no dilated or pulsating blood vessels such as when an aneurysm is present.

**Diagnosis.**—Primary erythermalgia is diagnosed on the history of reaction to heat and by elimination of other causes. Polycythemia, hypertension, and neurological lesions must be ruled out. Secondary erythermalgia may follow the lesions named above and have the symptoms of these diseases as well. Rarely, erythermalgia must be differentiated from Raynaud's disease. Hypersensitivity to temperature change may cause some symptoms similar to those observed in Raynaud's disease, especially when the patient is seen in warm surroundings. Blood counts and capillary microscopy help in the differentiation. A differential diagnosis between this lesion, acrocyanosis and primary Raynaud's disease is presented in Table 39.

**Treatment.**—1. *Primary Erythermalgia.*—Surgically, there is no treatment for primary erythermalgia. Underactivity of the sympathetic system so far has not been surgically corrected.

Medical treatment includes making the patient comfortable by wearin light clothing to avoid external heat and by exposing the affected extremi to a comfortable temperature Air-conditioned atmospheres help

In some patients there seems to be a definite psychic element responsib for the symptoms Most of the patients are nervous and hyperesthetic ar react violently to the symptoms of their condition In such cases th advice of a psychiatrist is indicated

TABLE 39 —DIFFERENTIAL DIAGNOSIS OF

| | *Raynaud's disease (primary)* | *Raynaud's syndrome (secondary)* | *Acrocyanosis* | *Erythromelalg* |
|---|---|---|---|---|
| Age | 10–50 | 20–50 | 20–40 | 30 50 |
| Sex | Females | Both | Females usual | Both |
| Onset | Color changes occas. acute | With claudication Color changes | Slow | Gradual |
| Site | Digits often bilateral | Toes or feet | Both hands | Lower extrem ties |
| Type of patient | Unstable | All types | Unstable | Frequently nervous |
| Pain | Mild aches | Severe | Occas. mild | Burning type worse with he |
| Claudication | Negative | Early severe | Negative | Negative |
| Trophic changes | Nail and finger tip | Early extensive | Severe sweating | Negative |
| Color changes | Pallor rubor cyanosis (attacks) | Rubor pallor severe | Mottling | Redness (attacks) |
| Reaction to cold | Initiates attack | Made worse | Yes | No |
| Edema | Negative | Negative | Yes | Rare |
| Phlebitis | Negative | With T.A O | Negative | Negative |
| Oscillometry and pulses | Normal | Reduced | Normal | Normal |
| Gangrene | Only in advanced | Yes | Only with frostbite | Negative |
| Temperature | Low in attack | Always low | Low in attack | High |
| X-ray | Negative | Sclerotic arteriogram block | Negative | Negative |
| Associated diseases | Nervous | Diabetes hypercholesteremia | Endocrine disturbance | Obesity |

To obtain relief some patients have had to move from a warm clima to a cooler one Vasoconstrictor drugs may help

2 *Secondary Erythermalgia* —When erythermalgia is secondary to an other disease, the treatment of the underlying lesion is primary Canc must be ruled out in all patients with erythermalgia.

## ERYTHEMA INDURATUM (NODULAR VASCULITIS)

Erythema induratum is a nodular type of vascular dermatologic lesion Although it occurs most often in conjunction with tuberculosis it is not o tuberculous origin in all cases as it was thought at one time When tu berculosis is not present no other microbe has been found to account fo the disease The condition occurs in the extremities most often in the leg

Erythema induratum results in a hypertrophy of the endothelium and fibroblastic cells in the lymph glands. There is marked endothelial proliferation in the blood and lymph vessels and there may be a secondary necrosis

The condition appears as nodules which are under the skin and are usually a reddish to a blue color

There is no specific surgical therapy for erythema induratum. It is of vascular surgical interest due to the vessel changes, the necrosis, and the necessity for biopsy, although localized lesions can be excised and skin grafted Biopsy is necessary to differentiate the lesions from erythema nodosum or tumors

The therapeutic use of the antituberculous drugs such as streptomycin and others (see page 151) or the antibiotics such as aureomycin may be considered

## ERYTHEMA NODOSUM

Erythema nodosum is an inflammation of the skin and subcutaneous tissues of the extremities It is of vascular surgical interest from the standpoint of diagnosis and biopsy

**Etiology.**—Erythema nodosum occurs most often in females It has been linked in many reports with rheumatic fever and it is most likely that the lesions are related to this condition Erythema nodosum has been seen in patients following the administration of sulfonamides, barbiturates, salicylates, and other drugs.[4] Some investigators have connected erythema nodosum with tuberculosis and diphtheria

**Symptoms.**—The nodules of erythema nodosum appear most commonly on the legs and arms, rarely on the head When present, the nodules are usually on the anterior surface The condition is self-limited and may last from two weeks to many weeks The lesions do not tend to suppurate

**Pathology.**—There is an accumulation of white blood cells, particularly the lymphocytes, around the blood vessels A secondary edema is present. Thrombosis is not usual, but it may occur Due to the pressure of the nodule, the fat may be displaced or necrosed

**Treatment.**—There is no specific treatment for erythema nodosum Because the lesions are so often associated with rheumatic fever, the same type of therapy is advocated, *i e*, salicylates The adrenal cortex drugs may be effective The possibility that the salicylates actually cause the lesions should be kept in mind If thrombosis of the veins results, therapy for this condition may require antithrombotic drugs or surgical ligation

Surgically, it may be necessary to perform a biopsy to make a diagnosis It is more important to eliminate malignancy from such nodules Erythema induratum can be differentiated pathologically

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Chapter

27

# SYMPATHECTOMY

## *Temporary and Permanent Interruption of the Sympathetic System Medical and Surgical Methods of Sympathectomy*

So important is the over and under activity of the sympathetic system to patients with vascular diseases that it is discussed separately in this chapter

The earliest reference to the sympathetic system was by Galen[23] in the second century Vesalius[94] discussed the ganglia and sympathetic plexus in 1543 Harvey[31] theorized regarding a connection between the circulation and the sympathetic system (1649) Claude Bernard in 1851 was close to the true picture in believing that the sympathetic system controlled metabolism and the body temperature John Hunter noted that blood vessels contracted when they were subjected to trauma The interruption of the sympathetic system basically is to eliminate the mechanism of spasm The part spasm plays in the arteriospastic syndromes is well established Its role in arterial occlusion has been recognized more recently The blood vessel under such circumstances may be likened to the modest lady who at the slightest insult, turns pale shrinks and runs away

The physiology of spasm is not well understood despite a great deal of investigative work. This is due to the difficulty of simulating exactly the same conditions in animals as occur in man Fear and pain so often enter the picture that any analogy drawn to man based on observations in animals may be erroneous The difficulty of transferring any conclusions from animal experimentation physiologically to man always is present but this applies in an even greater extent to the study of spasm (See Fig 146, page 454 )

**Theories** —Certain conclusions however are acceptable. A vasoconstriction may occur at the point of trauma or at a distance from it. This vasoconstriction may result from any number or types of stimuli or trauma

Physiologists are at variance as to what causes the spasm That there is an *autonomous* spasm at the cell level was proven by Landis'[44] work on the severed rabbit s ear as well as the investigations of Levenson and Essex[49] and the epochal work of Page[80] who demonstrated that a substance from the blood of a shocked dog would cause spasm in the vessels of a rabbit s ear

Whether this substance is histamine as advocated by Bach[6] Gopfert[28] and Lewis[58] or sympathin as postulated by White[99,100] or defibrinated

blood, a term used by Landis,[45] or unknown toxins produced in shock according to the beliefs of Page[60] and Chambers[12] or a change in vasomotor tone, as stated by Green,[28] probably is of little clinical importance. To simplify a complicated picture, our Clinic refers to the stimulator as "spastin."

That there is a nervous origin of vasoconstriction seems assured, according to the work of Phemister[62] and Barnes and Trueta.[7] The effect of sympathectomy also proves this point. It seems likely that there is some local ability of the vessel to "auto-constrict." This factor is not active unless there is a marked degree of trauma or stimulation. Some circulating stimulus acts directly or secondarily by changing the local vasotonia. It can act also reflexly through sympathetic synapses in the involved ganglia. Nervous phenomena or overstimulation of the sympathetic system may initiate it. That there is sympathetic control of the spasm factor, whether partial or complete, has been proven. An injury may cause the spasm. The trauma need not be external. For example, an intraluminal plaque in arteriosclerosis may initiate a reflex spasm in the vessel in which it occurs or in other vessels. Such a traumatic plaque also may cause a local autoconstriction, perhaps by changing the content of the blood at that point. The role of sludge and a slowed blood stream at the point of trauma as a cause for spasm is being investigated and requires further study. (See pages 178 and 612.)

Thus, a spasm reaction may occur whether the trauma is internal or external. The deposit of calcification on or in the intimal walls of arteriosclerotic vessels is an example of internal trauma. Afferent stimuli arise from the site of the trauma or occlusion. By a synapse with the sympathetic ganglia, these afferent stimuli produce efferent spastic stimuli to other parts of the same vessel and also to the collateral vessels. The transmitted mechanism may be chemical or neurologic.

The amount and effect of the spasm varies in different individuals and instances. The amount of spasm depends upon the stimulus, the degree and suddenness of the occlusion, the condition of the blood vessels (whether they are pathologic or normal) and the condition of the sympathetic system and its ability to respond.

The spasm is a normal physiologic reaction. There is a therapeutic reason for it. When a vessel receives a traumatic insult, hemorrhage may occur at that point. The spasm of that vessel helps limit this bleeding. In addition, if blood remains in these vessels, it will clot and thrombosis may occur. Spasm in the injured vessel and in the collateral vessels empties these vessels, thus retaining their patency for later use to establish the circulation around the blocked point.

**Types of Sympathetic Interruption.**—Sympathetic interruption may be temporary or permanent and may be produced medically or surgically.

I. Temporary Sympathetic Interruption
  A. Medical
    1. Intravenous general anesthesias such as ether.
    2. Intravenous local anesthesia such as procaine.
    3. Sympatholytic or adrenolytic drugs such as tetraethylammonium chloride, Priscoline (2 benzyl-4,5 imidazoline hydrochloride) and Dibenamine (N,N-dibenzyl beta chlorethylamine hydrochloride).

4 Others possibly vitamins etc

B Surgical

1 Sympathetic ganglion block

(a) Procaine or other anesthetic agent

(b) Procaine in oil or some other anesthetic in oil

(c) Alcohol

2 Spinal anesthesia

3 Epidural anesthesia

4 Periarterial sympathectomy

II Permanent Sympathetic Interruption

A Medical—no method known

B Surgical

1 Lumbar Sympathectomy

(a) Transperitoneal

(b) Extraperitoneal

2 Thoracic Sympathectomy

3 Thoracolumbar Sympathectomy

4 Cervical Sympathectomy (rarely used)

*Indications for Temporary Sympathetic Nerve Blocks*

(1) As test before sympathectomy

(2) As treatment

(a) To overcome arterial spasm

(b) Arterial occlusion

(c) Thrombitis (thrombophlebitis)

(d) Causalgia

(e) After arterial surgery

(f) Cerebral vascular thrombosis

(g) Possible pulmonary embolism

(h) Painful syndromes such as sub-deltoid bursitis

*Indications for Permanent Sympathectomy*

(1) Spastic diseases such as Raynaud's if medical measures are not effective

(2) Arterial occlusion

(a) of major vessels

(b) Poor collateral circulation

(c) Capillary bed failure

(3) Arterial occlusion without response to nerve block to improve collateral vessels and capillary circulation

(4) Chronic thrombitis patients selected i e thrombitis migraines

(5) Arterial occlusion i e injuries to arteries aneurysms embolisms where restoration of circulation is incomplete or inadequate

(6) Causalgia and Sudeck's atrophy

(7) Severe dysmenorrhea syndrome with presacral neurectomy

(8) Selected painful syndromes, such as pancreatic stones and chronic pancreatitis biliary dyskinesia (not amenable to direct attack)

(9) Hirschsprung's disease and other bowel atones (selected)

(10) Hyperhidrosis

(11) Ulcers of arterial origin selected See page 769 774

(12) Ulcers of venous origin selected See page 776

(13) Postthrombotic syndrome selected See page 781

(14) Patients after poliomyelitis, selected

(15) Residual changes after frostbite trench foot or immersion foot

(16) Erythromelalgia selected instances[39]

## TEMPORARY SYMPATHETIC NERVE BLOCKS

The sympathetic system may be interrupted temporarily by nerve blocks produced medically or surgically.

**Medical Nerve Blocks.**—Such nerve blocks produce only a temporary sympathectomy effect. Any drug ingested or injected which has sympatholytic or adrenolytic effect will act equally on all of the ganglia of the body. Thus, to obtain a local effect sufficient drug must be introduced to paralyze that ganglia. In using such drugs therefore, one must be prepared to accept the general effects of ganglia inactivity, namely, lowered blood pressure, slowed circulation, and, in some instances, vasomotor collapse. This latter will depend upon the degree of denervation produced. In patients in whom a lowered blood pressure and retarded blood flow is contraindicated, these generally-acting drugs should be administered cautiously, if at all. Such circulatory stasis may permit thrombosis in patients subject to coronary occlusion, cerebral vascular accidents or peripheral arterial occlusions. Where local effect is desired, such as in a compromised circulation of the limb, a local block is much safer.[16,29,69,74,92]

✓ (1) **Intravenous Ether.**—Ether (Katz[40]) may be given intravenously in a strength of 3 to 5 per cent in a 5 per cent glucose in normal saline solution. For practical purposes, 30 cc. of ether in 1000 cc. of saline solution makes a 2 or 3 per cent solution suitable for intravenous use. The solution is given usually for twelve to eighteen hours.

✓ (2) **Intravenous Procaine Hydrochloride (Novocain).**—This drug may be given intravenously in 0.1 per cent solution.[27] A suitable solution may be prepared by urgently agitating 5 cc. of a 20 per cent solution of novocain (1 gm.) in 1000 cc. of isotonic saline solution. This is injected with a "Flowrator" or by infusion drip with a clamp through a 19-gauge needle.

(3) **Tetraethylammonium Chloride.**—This drug is available commercially under the trade name "Etamon,"* in certain instances will block transmission of nerve impulses through the ganglia of both the sympathetic and parasympathetic areas in the autonomic nervous system.[7a] This interruption is temporary.

This drug is supplied in 20 cc. sterile vials containing 0.1 gm. of the drug per cubic centimeter or a total of 2 gm. The dosage must be individualized and is from 100 to 500 mg. (1 to 5 cc. given intravenously, but not to exceed 7 mg. per kilogram of body weight).

The drug can also be given intramuscularly, but at no time should the dosage exceed 20 mg. per kilogram of body weight when given by this route. The drug is not ✓adrenolytic but ✓sympatholytic in its action.

When injected intravenously or intramuscularly in sufficient dosage, tetraethylammonium chloride produces a temporary sympathetic nerve block.[2,7a,13] This apparently increases the peripheral blood flow in the capillary bed, as shown by some elevation of the skin temperature.

Untoward effects include weakness, hypotension, coldness, metallic taste, dilatation of the pupils and loss of ocular accomodation and vasomotor collapse. Artificial respiration, 2 or 3 drops of 1:1000 adrenalin and neostigmine help counteract a collapse.[13] Usually this is transient, but

* Etamon, Parke, Davis & Co., Detroit, Mich.

serious and even fatal shock has ensued It is suggested that if this substance is used, the patient be hospitalized where adequate measures to combat such an event are available [illegible]

(4) "**Priscoline**"*—Priscoline (2 benzyl-4 5 imidazoline hydrochloride) has been under experimental and clinical investigation since 1939, when it was reported by Hartmann and Isler[illegible] that injections of this substance decreased the blood pressure It was used particularly in South America and in Europe The first reports on its effects in peripheral vascular disease were made by Singer[illegible] in hypertension by Schroeder[illegible] and Kohlmayer[illegible] in Raynaud s disease and by Schnetz[illegible] in diabetic arteriosclerosis The early work in this country with this drug was performed by Grimson [illegible] Of the drugs available for sympatholytic or adrenolytic action, this one appears to be the least dangerous. It is similar to C-7337 (Regitine)

Priscoline apparently blocks the sympathetic motor pathways acting at the termination of the nerves in the smooth muscle This drug is adrenolytic in its action Priscoline can be used by mouth intramuscularly intravenously or intra-arterially Priscoline may have a histamine-like action in addition to its adrenolytic and sympatholytic properties When a small dose of 25 to 75 mg is given, there is a sensation not unlike that of fear and apprehension with a feeling of flushing or chilliness such as that which follows exposure to cold or fear The heart rate sometimes is elevated In those susceptible to the drug there may be nausea or vomiting In larger doses there may be dizziness sweating congestion of the nose headache, and audible peristalsis [illegible]

The use of Priscoline is suggested for the following

(*a*) *Raynaud's Syndrome* —Priscoline may help mild symptoms of Raynaud s syndrome

The dosage must be individualized to relieve the spasm in the individual Twenty five to 50 mg 4 times a day is usual

(*b*) *Occlusive Arterial Disease* —A large number of patients with occlusive arterial disease have been treated with Priscoline In those with very early changes and mild symptoms there has been some beneficial response. In the majority who have symptoms of sufficient intensity for them to consult a physician the complaints have not been ameliorated by this substance The excellent results of surgical sympathectomy and its low mortality rate in experienced hands make it the treatment of choice when it is combined with an excellent medical-control regimen

*Intra-arterial Priscoline* —This drug has been injected directly into the artery to register heat and flushing This reverses the pressor response to epinephrine.[71] The drug only partially inhibits the hypertension caused by norepinephrine There is a rise in pulse and fall in pressure but not as great a fall as in other drugs [73] There have been some remarkable and even dramatic results reported The danger of any intra-arterial manipulation in a limb where the blood supply already is threatened is a real one and may preclude routine use of this therapy If there is an indication it will be in the limb which appears lost and where the results after sympathectomy fail

* Priscoline Ciba Pharmaceutical Products, Inc Summit N J

*Dosage*—The dosage varies but is usually between 75 and 100 mg/daily

(c) *Hypertension*—In hypertension there has been no evidence that Priscoline will control the high blood pressure on a permanent basis

(d) *Causalgia*—These patients are difficult to treat because of the psychic factors involved The interpretation of the results of any form of therapy therefore must be subject to consideration of such psychic factors A trial with Priscoline is advocated

(e) *Other Uses*—This drug has been used in acute arterial occlusion[64] It has also been tried in acute cerebral accidents,[4,104] with the injection of 0 2 to 0 5 cc of a 25 mg per cc. solution of Priscoline into the carotid artery Such therapy has not been accepted universally

(5) **"Dibenamine."**—Dibenamine (N, N-dibenzyl-b-chloroethylamine hydrochloride) is one of the tertiary amines, structually related to nitrogen mustard (bis and tris chloroethylamines) Dibenamine by mouth causes nausea, vomiting, and burning in the epigastrium Its oral or local use is not practical

Dibenamine, used intravenously causes side reactions in 65 per cent of the patients[35] Extensive drowsiness and dizziness, sweating, nasal congestion, and tingling of the feet occur often Restlessness, irritation, palpitation, and pain at the point of injection are not unusual. There has been mental confusion Convulsions have been reported For these reasons, Dibenamine in its present form probably is not of clinical value.

The effective dosage appears to be a single intravenous injection of 4 to 6 mg per kilogram of body weight, or a total of 0.25 to 0 50 gm Dibenamine's reaction is not entirely adrenolytic but partially sympatholytic A reversal of the pressor response to epinephrine and nearly complete abolition of the pressor response to norepinephrine has been reported[21] Perspiration is blocked by the drug[32]

Further research may show that some derivative of this drug may have a therapeutic effect At present its use is not advocated.

(6) **Benzodioxane.**—This drug, when given in large doses, inhibits the norepinephrine hypertension The effect is very short The drug is supposed to neutralize circulating epinephrine and has been used for a test for the hypertension due to pheochromocytoma[24] This drug sometimes produces epinephrine reversal[21]

(7) **Hexamethonium (C-6).**—This substance is much like tetraethylammonium chloride and it increases the pressor effects of both epinephrine and norepinephrine It has had extensive clinical trial, particularly to produce hypotensive anesthesia (See chapter on Anesthesia )[19,21]

(8) **1-hydrozinophthalazine (C-5968[15,31,95]).**—In animals this drug partly blocks epinephrine and norepinephrine hypertension In humans it slightly inhibits norepinephrine but not epinephrine pressor response It does block the bradycardia caused by norepinephrine[21] It appears that the effects of these many so-called ganglion-blocking agents are inconsistent It has been suggested that this variation in response may be due to a blocking of the action of the effector cells at varying points in the chain

(9) **Other Substances with Questionable Sympathectomy Effect.**—Other substances have been reported to have some sympathectomy effect

(a) *Histidine in combination with ascorbic acid* has been described as having a vasodilatation effect.[illegible] The first report was enthusiastically hailed in the public press, but the experiences of other observers as well as our own have not been impressive [71,102,103]

(b) *Vitamin E*.—It has been known since 1942 that animals deficient in vitamin E are subject to degeneration in their vascular systems Evidence of this has been seen in the kidney, in the uterus and in the blood vessel walls particularly in patients with purpura

Shute and his collaborators[illegible] reported good results Ochsner[illegible] and his associates combined this substance with calcium to prevent thrombosis Our own study failed to confirm this theoretical improvement

Our Clinic has not been impressed with the value of alpha tocopherol (vitamin E) in any of the occlusive arterial diseases. It has been used in over 800 patients General hygienic instructions seemed to have similar effects The substance probably is valuable only if there is a vitamin E deficiency

(c) *Roniacol* *-3-pyridine-methanol has been reported to have sympatholytic effect. The patients develop a subjective warmth [illegible] A recent report showed no effect on blood pressure pulse rate or circulation in normal individuals [illegible]

(d) *Oranixon* (3-ortho-toloxy 1 2 propanediol) ** This substance was developed in 1946 in England to relieve muscular spasm of voluntary muscles It was found to have some analgesic properties similar to local anesthesia [illegible] This substance is a colorless crystalline solid which is rapidly metabolized and is excreted probably as a glucuronide [104] It depresses the hyperexcitability of spinal reflexes and thus is a spinal cord and brain stem depressant Its effect is similar locally to that of Procaine [illegible] Thus corneal anesthesia is achieved and the pain of muscle spasm is relieved [illegible] The substance was developed primarily for the treatment of tetanus and other severe disorders (hyperkinetic states) of the central nervous system

This drug which would relax smooth muscle reduces spasm and has an apparent local anesthetic effect was used in the treatment of arterial occlusive lesions It was hypothesized that the drug would have adrenolytic and sympatholytic effect. This medication has been administered to over 200 patients with apparent beneficial effect in the majority Claudication time has decreased and symptomatic relief has been effected in two groups These groups are the ones with minimal symptoms and those with signs of arterial occlusion who cannot be subjected to surgical sympathectomy It has been used in frostbite with relief of pain [76]

(e) *Others*.—Papaverine and nicotinic acid[77] are believed to cause a transient vasodilatation [illegible] A skin temperature rise of 1° to 2° C has been recorded but the response is variable Some flushing occurs but this may be an allergy or an antithesis. Papaverine is a derivative of opium The reports have been at variance [illegible] The effect allegedly is to relax smooth muscles Perhaps the best recommendation for the use of these drugs is that they do not hurt the patient if not used too long and in the proper dosage

* Hoffman-LaRoche, Inc Nutley N J

** Organon, Inc., Orange New Jersey

In summary it may be said at this time that there are drugs which may block the sympathetic system To be effective locally the dosages often must be so large as to produce shock and hypotension and thereby counteract their good effect

**Surgical Sympathetic Nerve Blocks.**—A sympathetic nerve block, carried out by the injection of an anesthetic solution into or in close proximity to a ganglion, will produce a sympathetic interruption for a variable length of time. This technic was first reported and used by Leriche,[48] and has been popularized in the last few years in this country by many others [58 97] Our Clinic has used and reported on it widely [70 71 74]

A sympathetic interruption so produced, while of a temporary nature, outlasts the anesthesia time Once the ganglion has been anesthetized, the spasm pathways, for which it is the synapse, are broken, and these pathways slowly re-establish themselves That the sympathetic effect may last as long as seven days is indicated by elevated potentiometer readings

Novocain in a 1 to 2 per cent solution is the usual anesthetic Five cubic centimeters are placed at each interspace

(1) **Lumbar Sympathetic Nerve Block (Pratt's Technic).** (See Fig 154)— There have been many technics developed for sympathetic nerve blocks The "best one" always is the one with which the operator is most familiar The following method has produced the sympathetic interruption most often and with the least discomfort to the patient in our Clinic's experience The patient is placed on his abdomen with a pillow between the crest of the ilium and the lower ribs to straighten out the lumbar curve of the spine The first, second, third, and fourth lumbar spines are marked with methylene blue or some other dye Other marks are placed exactly 4 cm lateral to the *intraspinous* space This point is selected because, in the lumbar area, the transverse processes are opposite the spinous processes With this technic the transverse processes are avoided Cadaver specimens indicate that 4 cm is approximately the correct distance to enable one to miss the body of the vertebrae despite the patient's physical size From the practical standpoint, the average spinal needle is 9 cm. long and approximately one-half of its length will be the correct 4 cm

The needle is introduced for a distance of 4 cm , or approximately half the length of the spinal needle At that time, the needle should be opposite the transverse process, but if the technic is followed carefully, the process will not be struck If the needle strikes the transverse process, it enters the periosteum and causes pain The patient invariably moves at this point, and his cooperation thereafter is difficult to secure

The needle then may be angled slightly toward the midline, to be certain that it is up against or in close proximity to the body of the vertebrae, and then it is introduced for another 4 cm The average-sized needle leaves approximately 1 cm protruding The other needles are introduced similarly Stylets are then withdrawn

Aspiration of each needle determines that the needle point is not in a large blood vessel or in the dura Five cc. of 2 per cent novocain are introduced at each point The needles are then withdrawn as one

The patient then lies on the side on which the solution was injected in order to pool the solution in that area A sympathetic effect generally occurs within a few minutes although in some if the technic has not been quite correct the response may not be elicited for fifteen minutes

*Other Technics* —(*a*) Labat [14] Ochsner[18] and others have advocated introducing the needles to strike the transverse process At this point the needle is elevated or depressed around the process and advanced to a point near the front of the vertebrae This method is adequate Objections to this technic have been discussed



Fig 151 —Typical Horner's syndrome after nerve blocking Note narrowing of the palpebral fissure constriction of the pupil conjunctivitis and ptosis of the lid

(*b*) One- or Two-Needles Technic —Either of the above methods may be utilized with the introduction of one needle at the 2nd lumbar area or two needles at the 2nd and 3rd Objections to this method are the occasional technical failures which occur when several of the ganglia are fused *i e* a large first ganglia only or a single ganglia at the 4th lumbar level A good sympathectomy effect after operation despite the failure of a block may be explained in this technical way Clinically we have demonstrated this many times Where four needles are used one will usually be in the psoas sulcus while a single needle reduces the chances technically four times

(2) **Thoracic Sympathetic Nerve Block Technic.**—The anterolateral approach is used in the technic for this block. The patient lies on his back with a pillow under the lower part of the neck. The head is turned to the opposite side. A 90-degree angle is made by a line through the supersternal notch and by a lateral line off the shoulder joint. Bisecting this 90-degree angle at 45 degrees, a wheal is made just above the clavicle.

The needle then is introduced at this 45-degree angle and pushed forward slowly, carefully and slightly cephelad until the body of the vertebra is felt. The needle can then be "stepped" slightly forward until it is near the anterior portion of the body of the vertebra, keeping the needle point against the body of the vertebra. The danger of transfixing a vessel is minimal if the needle is introduced slowly enough. At times, a transmitted pulsation can be felt when the needle point nears the carotid or vertebral artery. Ideally, the needle point is directly between these two.

The stylet is partly withdrawn from the needle and a drop of novocain is inserted at the needle base to eliminate the possibility that the needle point has entered the pleura. If there were aspiration of the novocain drop, *the stylet could be replaced without creating a pneumothorax*. After aspiration of the needle, between 5 and 15 cc. of 2 per cent novocain are then instilled.

The patient is observed carefully until the typical *Horner's Syndrome* (Fig. 151) is produced. This syndrome indicates that the stellate ganglion has been anesthetized. Horner's Syndrome consists of (1) a narrowing of the palpebral fissure, (2) a constriction of the pupil on the affected side, (3) a conjunctivitis on that side, and (4) ptosis of the eyelid. There is increase in warmth and absence of sweating. The presence of Horner's syndrome is an indication of a successful block. Once it has been observed, the needle can be withdrawn.

Certain technical dangers exist in doing the block. Technics which have for their purpose the anesthetizing of the second and third dorsal ganglia by angling the needle downward increase the danger of entering the pleura. We have witnessed the sudden collapse of a patient following a thoracic sympathetic nerve block as the result of the creation of a pneumothorax.

A needle occasionally has been introduced into one of the large blood vessels. *Little harm results if no anesthetic solution is injected.*

The needle has been known to enter the trachea or the esophagus when the operator is not experienced or does not use due care. In one instance to my knowledge, 10 cc. of novocain were injected directly into the esophagus, the patient regurgitating it through his mouth.

Each of these is a technical difficulty which can be prevented by care on the part of the operator.

*Other Technics.—Posterior Approach.*—This approach has been advocated by de Sousa-Pereira.[17] In this method the needle is introduced opposite the second dorsal vertebra on the affected side, approximately 3 cm. from the spinous process. It is advanced slowly until the vertebra is encountered. After aspiration the anesthetic solution is injected.[48, 63, 97]

*Other Dorsal Ganglia Blocks.*—At times the pain from angina pectoris is so severe as to be the initiator of coronary artery spasm or occlusion. In such instances their blockage may break this syndrome and even be lifesaving. The 2nd, 3rd, 4th and 5th dorsal ganglia are believed to be the cardiac

accelerator pathways. Paravertebral injections of these ganglia and occasionally the 6th have given relief to many patients. The technic is the same as for any paravertebral injection by the posterior route. Care is taken to avoid the pleura which is adherent to the vertebrae and ribs. The drop of procaine in the needle head as the stylet is slowly withdrawn helps materially to prevent a pneumothorax. Procaine is then injected. When the patient reports that there is pain relief, 1 to 2 cc. of absolute or 95 per cent alcohol are injected into each needle. The injection may have to be repeated. (See page 135.)

**Dangers of Thoracic Nerve Block.**—(*a*) *Injury to the Pleura.*—This is a definite possibility and in our experience occurs more often in the posterior than in the anterior approach. Pneumothorax and hydrothorax have been reported.[49,63,97] Collapse may follow such an accident. Death would result only if the pneumothorax was not recognized and treated by air aspiration.

(*b*) *Injury to a Blood Vessel.*—This complication can follow any injection near a large vessel, and in this instance the most likely ones would be the vertebral artery or vein. One case of cerebral air embolism after a stellate block has been reported.[3] Injections of anesthetic solutions or alcohol into the vessel could cause serious complications but technically are preventable.

(*c*) *Injury to the Spinal Cord or Nerves.*—The possibility of injecting solution directly into the dura exists. It is possible to introduce the needle through the intervertebral foramen but this is an unlikely complication. Subarachnoid injections can occur when an elongation of the dura extends surrounding the spinal nerve.[14] Nerve complications follow the injection into the nerves with a resulting neuritis. This occurs most often with the use of alcohol. In two patients epileptic convulsions have occurred during a block, again unrelated to the block itself.

(*d*) *Cardiac Death.*—Cardiac and respiratory paralysis have followed these injections. These are explained on the basis of an arrest or inhibition due to stimulation of the vagus nerve. The vago-vagal reflex with cardiac arrest is a real but rare complication. Prophylactically the use of atropine and the barbiturates helps to avoid its occurrence. These patients should never be left alone. If this complication develops it requires immediate active therapy. The patient's respiration should be continued by a rebreather anesthetic bag. If the heart is stopped, the chest must be opened *at once*—and the heart manually massaged. If the heart is fibrillating, an electric shock may defibrillate it. (See chapter on Cardiac Arrest for other measures.) Because of these complication possibilities, stellate block should be a hospital procedure.

*The Coronary Patient.*—A coincidental coronary or cerebral accident may be the cause of the fatality. In 1 patient in the author's series in which the thoracic ganglia were anesthetized for intractable angina, a death resulted. This was the fourth injection. The patient's pain was relieved on each occasion including the last one. One-half hour after the last injection, as the patient prepared to leave the clinic, he suddenly complained of chest pain and died. In this patient, without question, the death was coincidental. In a second patient a coronary occlusion occurred following the raising of a wheal on the skin preparatory to an injection. These patients are in imminent danger of death at any time. As a safeguard, the

**Criteria for Sympathectomy for Occlusive Arterial Disease.**—The best results from sympathectomy for patients with occlusive arterial disease will be obtained in the following five groups

(A) *Patients Who Will Respond Best to Sympathectomy.*—

(1) Those who have discontinued smoking completely and permanently

(2) Those without major gangrene or advanced infection

(3) Those in whom spasm is a factor, as shown by the skin temperature rise of 3° to 12° when a sympathetic nerve block is performed

(4) Some patients will have an excellent result from sympathectomy despite a negative response to a block In these patients, the block fails for technical reasons or because of arborization of the chain which sympathectomy removes

(5) Those with advanced occlusion in whom a transmetatarsal or a below-the-knee amputation is desired instead of a thigh amputation

If a sympathectomy is performed in these five selected groups of patients, circulation in the capillary bed and in the skin will increase This is an important consideration, because gangrene, when it occurs, begins in the skin

(B) *Other Patients Who May Respond to Sympathectomy* —

(1) Patients who have had amputations for occlusive arterial disease Since this disease is generalized, similar trouble is to be expected on the opposite side It is our practice to advise sympathectomy on the side of the remaining limb In no instance (in over 200 sympathectomies) have we seen such an operation precipitate gangrene as has been reported by others

(2) Patients in whom there is question of the amputation stump healing

(3) Patients with mild occlusive arterial disease whose occupation subjects them to environmental or traumatic dangers While an effort should be made to change the occupation of such individuals, in some, training or age makes such rehabilitation impossible

(4) As a supplement to amputation below the knee or through the foot Surgical sympathectomy can be performed in twenty minutes and we use it in many cases as an adjunct to an amputation at a lower level to insure healing It requires judgment to select patients for such a procedure but we have had to re-amputate only 2 of 20 so treated

In recent years, there has been some controversial discussions as to the effect of sympathectomy on the blood supply to the muscle Claims that there is reduced arterial supply in the calf muscles after sympathectomy[20] have not been proven Efforts to prove the reduction in blood flow in the muscle by the rate of removal of radioactive sodium are inconclusive since diseased vessels may be slow in picking up such a substance while normal tissue fluids may be carried away rapidly Unless the muscle blood vessels are entirely different than those in the skin, their blood supply also increases The number of patients in whom claudication is reduced bears this out Gangrene begins in the skin, and the capillary supply to the skin without question is increased after sympathectomy

## LUMBAR SYMPATHECTOMY

**Anatomy of the Lumbar Sympathetic System.**—The anatomy of the lumbar sympathetic chain varies considerably The difficulty in its identi-

fication is known by all who undertake this operation for the first time. Dissections on cadavers have brought the peculiarities of the chain's distribution to light. It is important that the trunk be divided below the level of sympathetic outflow from the spinal nerves. Sheehan[47] described this as the second lumbar segment. A line connecting the iliac crest passes through the 4th lumbar vertebrae in the majority of men. In the female, however, this line may vary from the 3rd disc to the 5th vertebral body. This may lead to technical errors. An *x* ray prior to operation with the patient in the position in which he will be operated is indicated if there is question as to the level. The sympathetic chain in the lumbar area is on the anterolateral surface of the spine. The chain may thin out to a fine thread and this has been found to be true most often between the 12th dorsal and the 1st lumbar and between the 3rd and 4th lumbar ganglion. The ganglia in the lumbar area vary and often fuse. The rami communicans may be from one to three or four for each of the lumbar nerves. These usually arise from the same ganglion level but occasionally develop from ganglia at other levels. Recent anatomical dissections confirm the fact that these communicating fibers from the 12th thoracic and 1st and 2nd lumbar most often pass cephalad while those from the 3rd, 4th and 5th are directed caudad.[74] The 1st lumbar ganglion supplies the area around the knee and above it and may be of value in the production of collateral vessels. At times a middle diaphragmatic crus may hide the sympathetic trunk. Some accessory sympathetic pathways exist. This is responsible for some sympathectomy failures but denervation depends upon the degree of completeness of the sympathetic excision. The genitofemoral nerve has some connections with the lumbar sympathetic trunk and may be of importance in failures. In one-half of the patients the number of ganglia are four but these vary on the two sides. There seems to be no doubt but what there are communications between the right and left chains.[11,42] While most textbooks state that the removal of the first lumbar ganglia bilaterally results in a permanent loss of male sex ejaculation, this is not certain or consistent. Twenty four of Smithwick's patients reported loss of erection and 25 per cent loss of ejaculation.[91] One of our patients after bilateral extensive thoracolumbar sympathectomy for hypertension has had children. The possibility that L-4 and 5 are postganglionic and therefore their removal sensitizes the patient to adrenalin has not been proven.

The sympathetic area contains other important structures such as the peritoneum and its contents, nerves, the ureter, the kidney, the genital vessels and of course the vena cava, the aorta and their branches. The lumbar veins require special note and attention since their rupture causes many of the serious accidents reported.

(1) **Transperitoneal Approach.**—Where the sympathetics of both lower extremities are to be interrupted, the transperitoneal approach saves one operation. The patient's general condition or previous operative intervention may preclude its use. If the patient's general status is not too good, the extraperitoneal approach should be elected. Spinal anesthesia is selected. With present surgical technics the abdomen can be opened as safely as the retroperitoneal space. There should be no greater incidence of ileus than from the retroperitoneal approach. With early ambulation

much of the accompanying ileus and other complications can be prevented. A bilateral lumbar sympathectomy may cause a hypotension In those patients who have a low blood pressure, have had a coronary occlusion or a cerebral vascular accident, or in the aged, the extraperitoneal route with unilateral sympathectomy is advocated

**Operative Technic for Transperitoneal Lumbar Sympathectomy.**—The patient is placed on a tilt-type operating table with his back over the

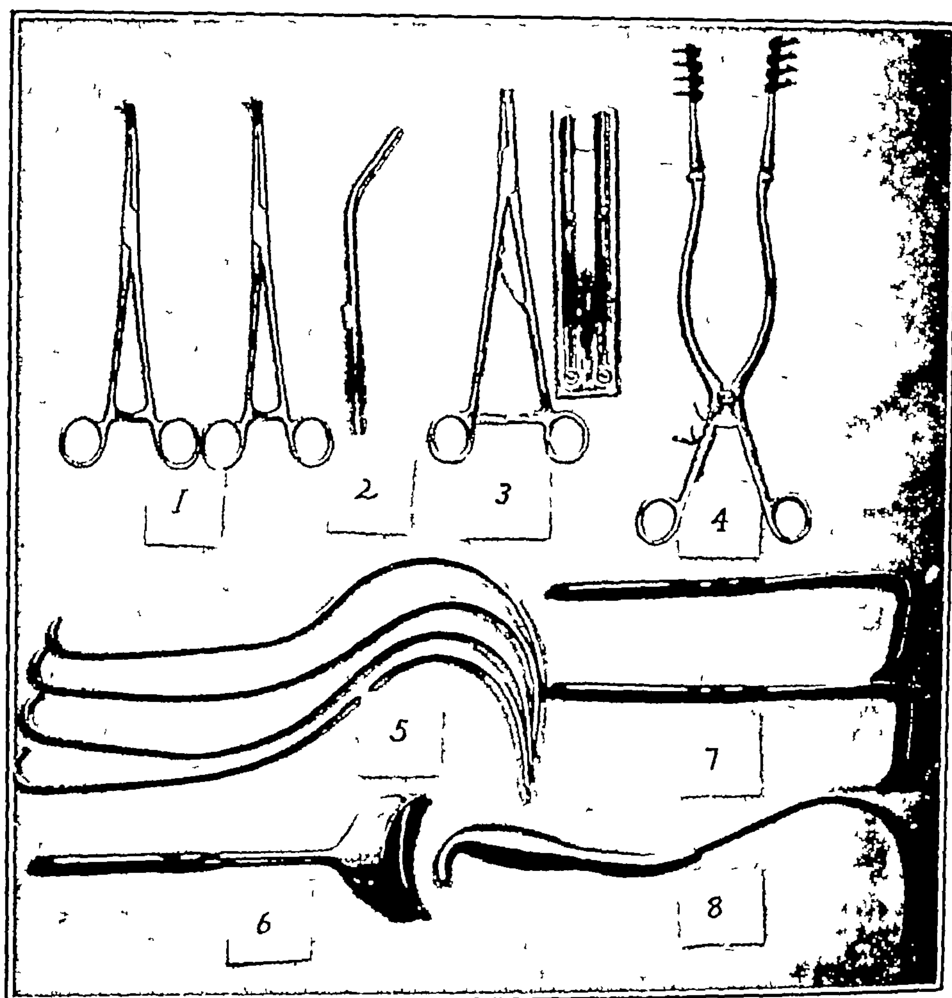



FIG 152 —Special instruments used in sympathectomy 1 Gallbladder-type clamps 2 Suction 3 Brain clip apparatus 4 Self-retaining retractors 5 Deavers, 3 sizes 6 Abdominal retractors 7 Abdominal retractors 8 Harrington retractor

lumbar rest or lift, which later can be sufficiently elevated to bring the lumbar spine close to the abdominal wall A vertical or muscle-splitting periumbilical incision 4 inches long is made The Trendelenberg position will help in packing off the intestines All the small bowel and its mesentery should be elevated with warm packs It is important not to leave a loop of small bowel in the midline to avoid an incision in its mesentery. The cecum and sigmoid are displaced to either side by an abdominal pad

The mesentery of the cecum and sigmoid is not disturbed These organs need not be mobilized or dissected, other than to pack them off

to their respective sides With the bowel elevated a midline incision is made in the retroperitoneum from L I to L IV, this incision paralleling the *medial border* of the *aorta* Tilting the table sideways and elevating the

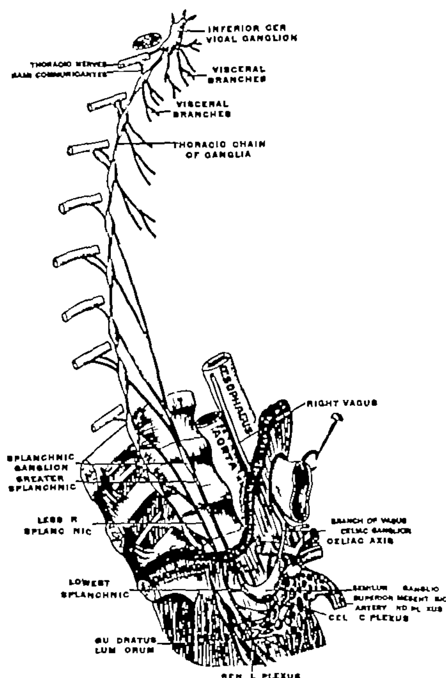



FIG 153 —Anatomy of the sympathetic cord and splanchnic nerve on the right side

kidney rest helps the exposure. Both chains can be removed through this single retroperitoneal incision

On the right side the vena cava is partly rolled and displaced medially and the sympathetic chain located by palpation The chain is in the sulcus between the psoas muscle and the body of the vertebra adherent to the

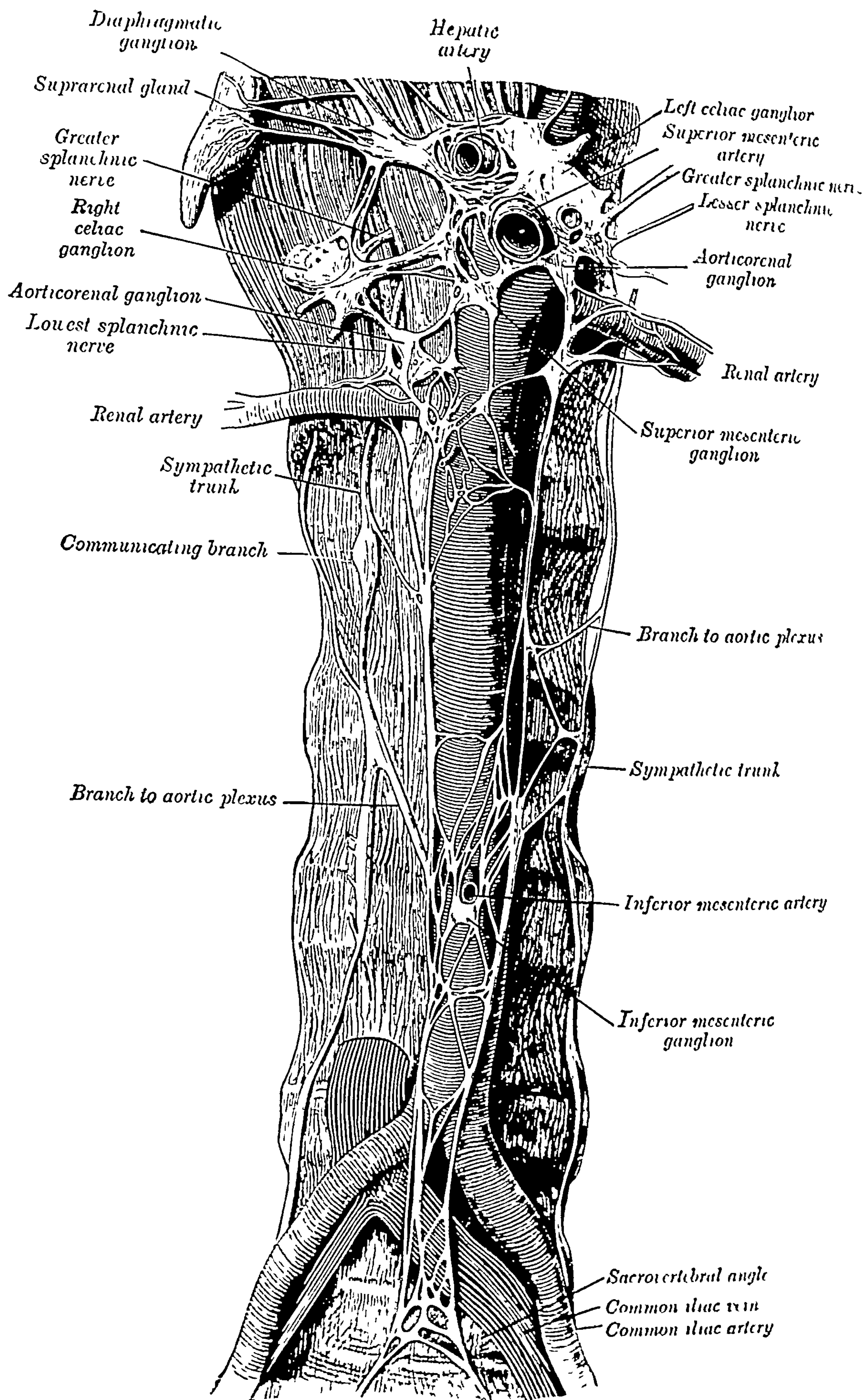



Fig. 154.—Lumbar sympathetic trunk. Note the number of communicating branches to the aortic plexus. Necessity for a complete denervation to restrict sympathetic effect is emphasized. Each branch from the chain should be avulsed.

vertebra and directly beneath the vena cava The chain has a characteristic feel similar to that of the vas deferens A moistened mounted peanut sponge forceps makes an excellent retractor to displace the vena cava at *this stage without injuring it* The chain is grasped with a clamp to keep it on tension during its dissection from the surrounding tissues The cord

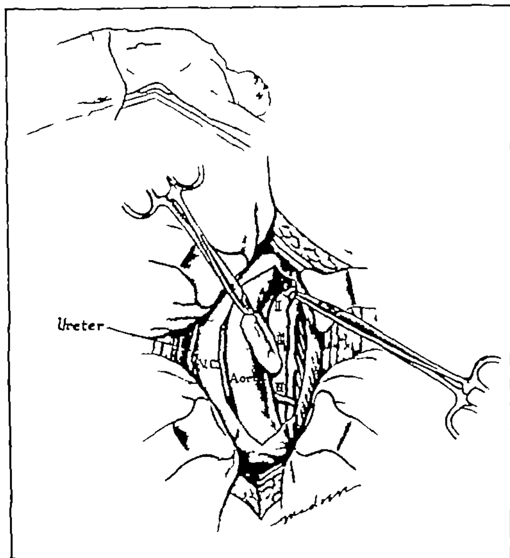



FIG 155 —Transperitoneal lumbar sympathectomy Technic of transperitoneal approach for lumbar sympathectomy The viscera has been packed into the upper abdomen. The retroperitoneum has been opened in the midline. The aorta and the vena cava are rolled medially in turn so that the lumbar chain can be removed. Insert shows patient s position on operating table.

from L II to L IV routinely is resected It will be noted generally that the connecting fibers of the chain point caudad on the third and fourth ganglia and cephalad at the first and second ganglia

As the ganglion of L I is partly covered by the diaphragm it is removed by exeresis similar to that used in phrenic nerve interruptions The chain is grasped by a forceps and pulled down in the direction of the vertebra *not*

toward the wound surface. A larger segment of the chain can be removed in this way. At times, many branches of the chain—sites of possible future anastomoses—come away with the chain and ganglia. Because the chain tends to break up into many branches below L III, the lower end is exeresed in a similar fashion.

The lumbar veins cross the chain and ganglia at right angles. These may be very large, especially if there has been a vein blockage previously. The cord can be teased under each of these. If there is annoying hemorrhage from a lumbar vein, it is best controlled with a brain clip. One should *not* try to clamp and tie these vessels as they are fragile, valveless, tear readily and may lacerate into the vena cava. The heavy lumbar muscles help to control this bleeding.

On the aortic side, the chain is slightly more lateral. The dissection is somewhat more difficult in this area due to the aortic glands and their blood vessels.

The sympathetic chain lies on and is adherent to the vertebræ close to the large blood vessels. The genitocrural nerve, other lumbar nerves, the ureter, fascial strips, and blood vessels, all have been mistaken for the chain. If all of the structures of the abdomen were avulsed en masse, the sympathetic chain would still remain attached to the vertebræ. Any structure therefore that is freely movable or lateral cannot be the sympathetic chain. The "tell-tale" ganglionic swelling should be identified.

After hemostasis is secured, the retroperitoneum is united with No. 0 plain catgut suture and the bowel is replaced in its normal position. A routine appendectomy is suggested. Manipulation of the bowel may initiate an appendicitis, and a secondary operation thus can be prevented. The outer peritoneum and the abdominal wall are closed in the usual fashion. We use interrupted steel wire in this anterior abdominal closure to permit ambulation the same day.[66,67] In our experience, this early ambulation has reduced the postoperative pulmonary and venous complications by 85 per cent.

(2) **Extraperitoneal Lumbar Sympathectomy.**—The lateral abdominal approach routinely has replaced all other operations. The two sides are done a few days apart unless the transperitoneal operation with bilateral sympathectomy is used.

(A) *Position.*—The patient is placed partly on his side, with the operation side elevated. He is supported by a sand bag under the hip and the shoulder. The under leg is bent at the knee and the leg on the operative side kept straight, the two legs being separated by a pillow. This keeps the psoas muscle on tension, rather than relaxing it. Relaxation of the psoas permits it to cover the chain and ganglia. The arm on the operated side is elevated and grasps the upper corner of the opposite side of the operating table. This position lifts the lower ribs out of the operative field and extends the distance between the crest of the ilium and the thoracic cage. The table may be broken at the lumbar level, tilted when necessary and the lumbar rest elevated to further improve the position. We anchor the patient with a strip of 3-inch adhesive, which extends from the bottom of the operating table, across the flexed knee and the greater trochanter of the operative side, again down to the bottom of the operating table.

(B) *Anesthesia* —Spinal anesthesia is an ideal agent for this operation. The anesthetic level should be to the 6th dorsal vertebrae. In operations on patients with vascular disease intravenous anesthetics should be avoided if possible. Patients who have diseases of their blood vessels frequently associate exacerbations or complications with a needle inserted into a blood vessel. In addition many patients with arterial disease have venous complications which may be excited by a vena puncture. This

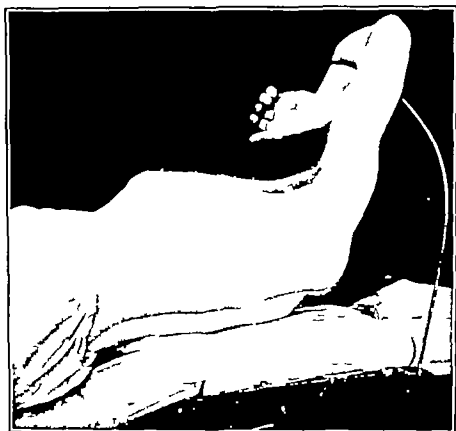


FIG 156 —1 Position of patient for retroperitoneal sympathectomy. Operative site raised by sandbag to 45° to permit viscera to "fall away" from the surgeon.
2 Leg on the operative side straight to keep psoas on tension. Opposite leg bent at knee under this leg with a pillow between the legs.
3. Arm of operative side swung across and above operating table to pull lower ribs from wound area.
4 Table broken to extend space between ribs and crest of ilium.
5 Patient anchored on table by adhesive tape. When correct this position brings vertebrae directly beneath incision side without intervening viscera.

must be explained to the anesthesiologist who with his modern technic is anxious to avoid any discomfort to the patient. If this is not done the patient may have an intravenous injection running before the surgeon realizes it.

(C) *Operative Technic* —A 3-inch transverse incision is made on the lateral side of the abdomen 2 inches above the iliac crest at the level of the second lumbar vertebra. (See Fig 157.)

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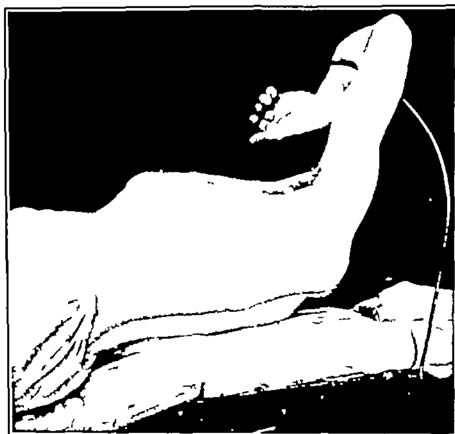


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(C) *Operative Technic* —A 3-inch transverse incision is made on the lateral side of the abdomen 2 inches above the iliac crest at the level of the second lumbar vertebra (See Fig 157 )

The external oblique muscle is divided in the line of its fibers, as is the internal oblique The *transversalis* muscle is split at a level in the wound so that one will be approximately at L II when the vertebra is reached If this muscle is divided *too low*, it makes the division of the upper ganglia difficult The transversalis muscle should be split well lateral to the rectus muscle to avoid injuring the peritoneum, as the peritoneum is adherent to the posterior sheath of the rectus muscle

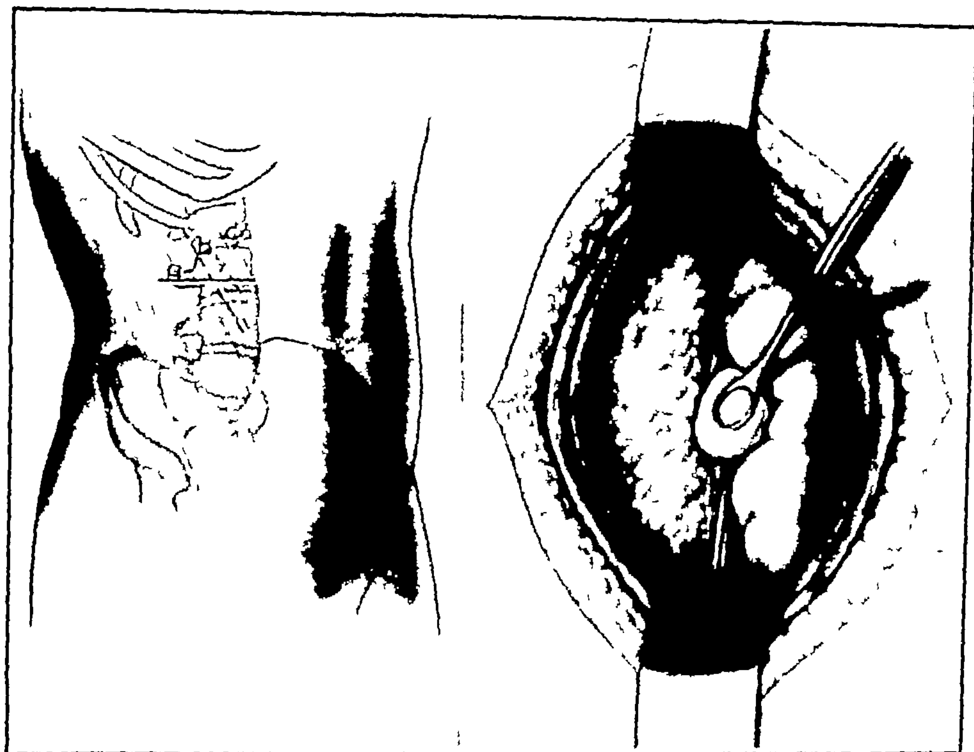


Fig 157 Fig 158

Fig 157 —Extraperitoneal lumbar sympathectomy Site of incision and the line of cleavage of the various muscle layers in extraperitoneal sympathectomy The skin incision is in *line of skin cleavage* (*a*) The external oblique is divided in the line of fibers—muscle runs like pants pockets (*b*) The internal oblique is divided in an oblique fashion (*c*) The transversalis is divided transversely as in *a*

Fig 158 —Lumbar sympathectomy Viscera pushed forward by a mounted sponge forceps to expose the psoas muscle Note the retroperitoneal and perirenal fat posteriorly

The peritoneum and its contents are pushed forward with *mounted* sponge forceps, the line of cleavage being anterior to the kidney and the psoas muscle The psoas muscle and its fascia are exposed The peritoneum is stripped from its anterior surface until the sulcus between the psoas muscle and the vertebra is *reached* The fascia over the sulcus is divided

On the *right* side, it is not necessary to denude or even mobilize the vena cava This vessel and its *surrounding* fat are pushed forward The sympathetic cord is identified by rolling it under the finger against the vertebrae Harrington and Deaver *retractors* will help isolate the chain, which may be held tense by a clamp or a silk ligature during its dissection with nerve hooks (See Fig 159)

The chain is freed from its vertebral integument. The so-called 'fool's sympathetics,' the ilioinguinal and the genitofemoral nerves, are more lateral. The ureter has been mistaken for the chain. The chain and ganglia from L II to L IV and often L I are removed routinely as described under the transperitoneal approach on page 502.

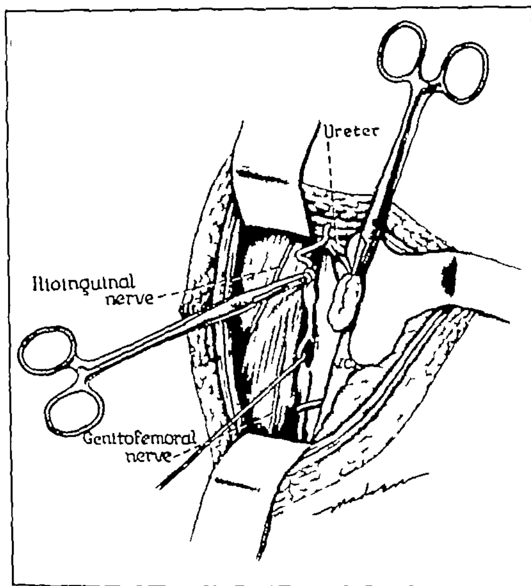



Fig 150 — The sympathetic chain is removed by excision. A large segment of the cord is thus removed. The effect of the sympathectomy varies directly with the completeness of the procedure.

On the *left* side the operative procedure is similar. The aortic glands make dissection slightly more difficult on this side. Brain clips help control any bleeding deep in the wound. After the chain is removed the muscles are permitted to fall together. The transversalis muscle is united with care to include the transversalis fascia. Hernia may occur if this precaution is not taken. The same sutures are inserted in the internal and external oblique muscles. Interrupted wire sutures (#32) permit ambulation the

same day The wires are buried by a subcutaneous fine catgut suture Number 36 steel wire is used for the skin

**Gangrene after Lumbar Sympathectomy.**—There have been some reports in which conclusions were drawn that lumbar sympathectomy precipitated gangrene We believe that in such patients when sympathectomy was performed gangrene already was inevitable [72] In these patients, sympathectomy did not precipitate the gangrene, it merely did not stop it It has been claimed that the release of the sympathetic control of the juxtamotor system shunts blood through arteriovenous circuits and decreases the blood flow in the capillary bed [5,20] This has not been proven and, clinically, is contrary to all observations Smithwick[92] mentions that "most if not all bad results are due to improper selection of cases and that the vast majority of patients who fail to improve or get worse following sympathectomy are those who have extensive main vessel obliteration without an adequate collateral circulation " Ochsner[57] calls it "the gangrene which develops despite sympathectomy "

When sympathectomy is performed on patients in whom the outcome is doubtful, the patient, the patient's family and physician should be so informed, so that the procedure in itself will not be given discredit should the changes in the limb already be irreversible The sympathetic procedure should not be delayed too long

In some patients, even though we thought the gangrenous process was irreversible, we have performed a sympathectomy at the patient's (or the doctor's) insistence In many of these instances the gangrenous process was forestalled In others, however, gangrene continued with no change.

In a review of the last 200 lumbar sympathectomies performed for arterial occlusion, there is no instance in which gangrene was precipitated by the operation itself [71] In 13 of the 200, however, gangrene did develop subsequently, and amputation had to be performed In 2 of these 13 the sympathectomy was performed with the amputation to insure better healing in the stump where continued sloughing was feared In 2 others the pathologic diagnosis of sympathetic tissue was not confirmed, and the technic of the resident had to be questioned In the other 9 it was felt that the decision as to survival of the part had been decided adversely by Nature already In several instances, it was possible to perform an amputation below the knee with satisfactory healing, although it is certain it would not have been possible without the sympathectomy (See Fig. 151 )

In this group of 200 there were 2 deaths, although these deaths were not attributable to the operation One patient was an advanced arteriosclerotic, a confirmed and heavy consumer of alcohol and had had several attacks of so-called "liver trouble " After the operation, he developed a renal and hepatic insufficiency with a final hepatic death The sympathectomy wound was entirely healed and there was no complication from a surgical standpoint It had to be considered a sympathectomy death inasmuch as it occurred within a few weeks after the operation The second death was due to an occlusion at the bifurcation of the aorta (See Fig 57 )

*Possible Injury to Vessels During Sympathectomy* In any discussion of gangrene following sympathectomy, it must be remembered that in per-

forming a sympathectomy one is in very close proximity to the aorta and other major blood vessels In some of these arteries there are large arteriosclerotic deposits and plaques and roughness or retractor pressure on the aorta or some of its branches may loosen a plaque or calcification The result is hemorrhage in the wall followed by occlusion or peripheral embolism Thus this added trauma may be the actual cause for the so-called precipitated gangrene rather than the sympathectomy

**Technical Points in Lumbar Sympathectomy** —The physiology of the sympathetic system and the part of the body supplied exactly by each ganglion is not known This is apparent to all who are doing this type of work. While the investigations of Smithwick[64] and White[89] detailed to some extent which ganglia contain ganglion cells we still do not know for certain when a sympathectomy is preganglionic and when it is post ganglionic

**Completeness of the Sympathectomy** —For the occlusive arterial disease the degree of improvement in our experience follows closely the thorough ness of the sympathectomy For this reason we do not believe that mere division of the second and third lumbar ganglia and sympathetic cord is sufficient to denervate the lower extremities

Many times the lumbar chain and ganglia are fused and only one ganglia performs the function of the usual four The tendency of the sympathetic system to reactivate itself is well known See page 154 In sympathectomy the ganglion and chain should be identified and dissected free as far as it is possible to do so visually The chain can be wound on a hemostat and exercised by pulling down and then up Often a long tailed nerve end is removed from either end by this method

Many small branches that might have re-established themselves will be removed There has been no evidence that removing the ganglion in this way below the fourth lumbar vertebra has produced the effect evidenced after a postganglionic operation

**Postoperative Complications** — 1 *Ileus* —Postoperatively there is a mild abdominal distension This is an adynamic ileus which accompanies all retroperitoneal operations or injuries This usually subsides in a day or two If persistent it may be treated by an indwelling duodenal catheter with suction syphonage [74] Stringent efforts to overcome this distention with such drugs as pituitrin and pitressin are not physiological This ileus is Nature s effort to splint an injured area and when no longer needed it will subside spontaneously

2 *Effect on Sex Function* —In bilateral lumbar sympathectomy where L I is removed on both sides sterility may result (approximately 30 per cent) The power of ejaculation may be lost if the sympathetic fibers of the first lumbar ganglion are divided completely and permanently on both sides but other sexual characteristics in the male including potency are not changed Many who have temporarily lost ejaculation recover this power later possibly due to some other levels taking over the innervation or due to partial re-establishment of the sympathetic control Apparently no changes occur in the female

3 *Neuritis* —An annoying neuritis may be a complication after lumbar sympathectomy This occurs along the anterior thigh The ventral surface

of the thigh is supplied by the branches of the 1st, 2nd and 3rd lumbar nerves These may be traumatized during resection of the ganglia. In our experience this occurs in 5 to 10 per cent of the patients Others have reported as high an incidence as 55 per cent. The condition develops approximately one week or more after the operation and in general is self-limited (six weeks). Large doses of vitamin $B_1$ and $B_{12}$ intramuscularly and by mouth and an intravenous injection of sodium salicylate and sodium iodide, grains 20, in the treatment of this annoying complication, ameliorate the complaint Sedatives and reassurance are essential

4 *The Prevention and Control of Hemorrhage* —The small lumbar veins run at right angles over the spinal cord and ganglia In certain instances, particularly after a patient has had phlebitis, these veins may be of a large size Since they open directly into the vena cava, they are potentially dangerous from the hemorrhage standpoint

After experience, it will be found that laceration of these veins can be avoided in doing the exeresis by pulling directly down and directly up along and in the direction of the vertebral column In certain instances, the cord can be grasped above and below such a large vein and the chain divided and slipped along under the vessel

If these veins are torn, the hemorrhage may be annoying but not particularly dangerous, provided the efforts to control it do not increase the damage Bleeding can be controlled usually without difficulty by brain clips Bleeding vessels should not be clamped or ligated Efforts to ligate such vessels in this deep area often cause further tearing, as suturing or ligating in such a wound and close to the vena cava is technically difficult

With a mild suction a brain clip can be applied to the bleeding point Even arteries can be controlled adequately by these brain clips applied at or near the hemorrhage point Should the vessel be torn so close to the vena cava that it is difficult to apply a brain clip, pressure on the area for a short time may stop the bleeding, or a small piece of psoas muscle held against the point by a clip usually will control it Muscles in this area are so large that when the retractors are pulled out, these muscles are hemostatic in themselves

In all such operations only one patient has developed a postoperative hematoma The viscera in itself is partly hemostatic when it is replaced If necessary, some gelatine substance ("Oxycel," or "gelfoam," see page 326) can be left as a hemostatic pack The surgeon is again cautioned against too active efforts to stop minimal bleeding in this area It is best to let Nature have a chance If bleeding is uncontrollable, the safest measure is a pack The author has amputated two extremities for gangrene where the surgeon in his efforts to stop bleeding finally tied a major artery

## THORACIC SYMPATHECTOMY

Our conception of the sympathetic innervation is not complete and still is subject to change This lack of knowledge applies more in the upper than in the lower extremities The actual function of each ganglion and the overlapping of these functions has made the correlation of the effect of sympathectomy in the thoracic and cervical regions uncertain.

Certain facts are known about the thoracic sympathetic system however and these are (1) The greatest sympathetic control of the upper extremities is exerted through the second and third dorsal ganglia

(2) The fifth the fourth the third and possibly the second have cardiac acceleration fibers in their ganglia

(3) In most instances the cervical sympathetic fibers do not have a sympathetic effect in the upper extremities

(4) The stellate ganglion a combination of the seventh cervical and first thoracic ganglia in most instances does not pass sympathetic innervations to the upper extremities

(5) At other times this stellate ganglion does permit transmission of a sympathetic innervation to the upper extremities

(6) At times after thoracic sympathectomy the stellate ganglion may take over some of these pathways to the upper extremities

(7) The greater lesser and least splanchnic chains have their origin in the ganglion beginning at D VI

Sympathetic denervation of the upper extremities has not been as satisfactory as it is in the lower extremities Originally a stellate ganglionectomy was the operation advocated This procedure denervated the upper extremity for a time After six to nine months however the reaction to the stimulation which caused the spastic syndrome in the upper extremity returned and in many instances was more active than before It appeared that the parts became oversensitive to the adrenalin sympathin or as we called it spastin which was liberated into the blood stream as the result of the emotional thermal or traumatic stimulant This reaction appeared to be similar to that which occurs in a postganglionic denervation Smithwick and White[69] devised an operation which they considered preganglionic in type Their operation is based upon the deduction that if the ganglionic cells are not removed the operation is preganglionic In the upper thoracic area they believed the ganglionic cells are in the ganglia in contradistinction to their situation in the lumbar area where they are not so placed

Their operation therefore consisted of division of the thoracic sympathetic chain below D III removal of all of the communicating branches between the ganglia and the vertebræ resection of a segment of the 2nd and 3rd intercostal nerves and rep'acement of the sympathetic chain outside the thorax without removal of the ganglia White wrapped the chain segment in tantalum foil and Smithwick enfolded it in a piece of fine mesh silk cloth Thus one end of the sympathetic chain was in the thorax and the other end was outside of the thoracic cage

Later we performed a similar excision but sutured the upper end including the ganglia into the intercostal muscle thus avoiding the inclusion of a foreign body [70]

In the majority of patients this operation was successful although technically difficult There remained however a definite number of patients operated in the above manner who continued to have symptoms of spasm and had cold hands

There were efforts to improve the technic The anterior approach was tried many times sometimes successfully sometimes not A return to

ganglionectomy was then attempted with removal of the 2nd and 3rd ganglia and chain and at times the 4th In most instances this operation was effectual, indicating the ganglionic cells probably are not in the thoracic ganglion area

It is admitted, however, that frequently the results were not satisfactory despite the operation performed We now use the open thoracotomy technic for thoracic sympathectomy (see pages 516 and 519) The operations are presented below

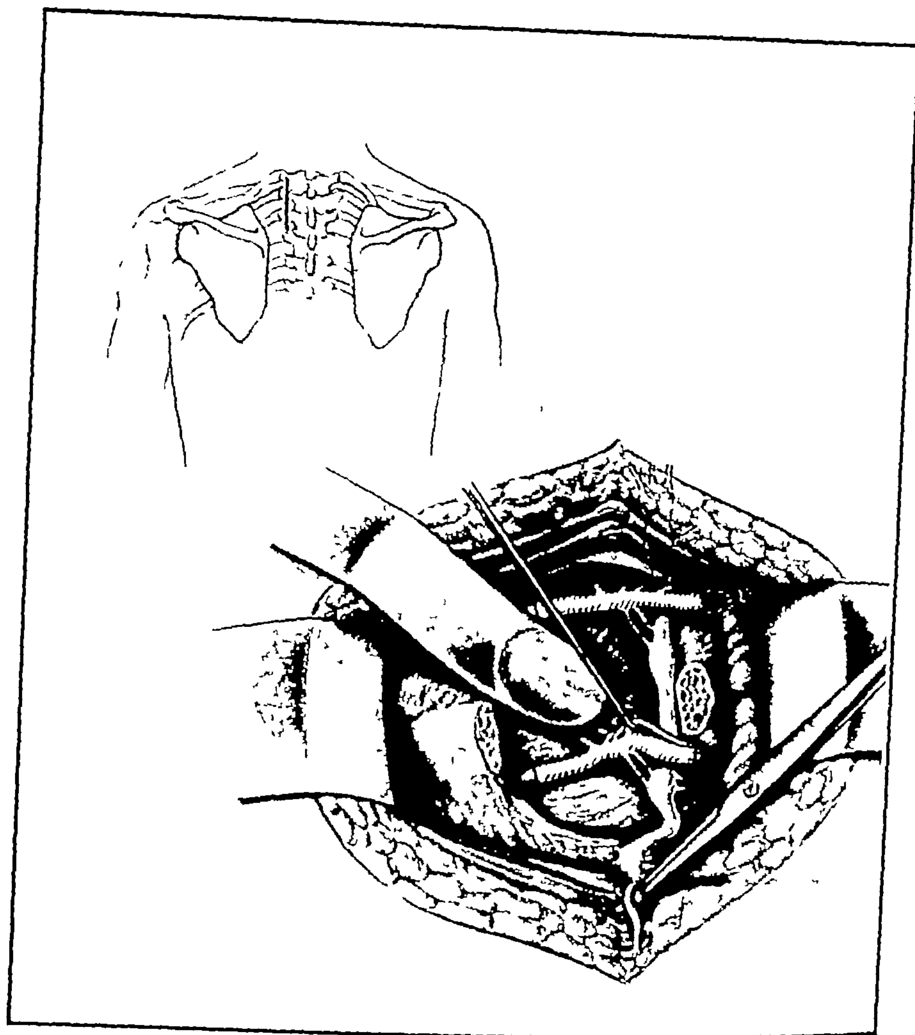


FIG 160 —Thoracic Sympathectomy Modified Smithwick-White operation Insert shows line of incision for thoracic sympathectomy Second view shows the third rib and transverse process having been resected The second and third intercostal nerves are identified The sympathetic nerve and chain are dissected and all branches from the chain to the 2nd and 3rd nerves and to the spinal cord are divided The brain spoon is retracting the pleura The sympathetic chain is divided below the third dorsal level and its connections to the cord and the somatic nerves resected, but the chain and ganglia *are not* removed

**Operative Technic of Thoracic Sympathectomy.**—(1) *Smithwick-White Modified Technic* —The posterior approach is selected for thoracic sympathectomy The anesthesia should be endotrachial to avoid the danger of a tension pneumothorax should the pleura be injured A vertical or oblique incision, beginning at the first rib and extending to the fourth rib, is made approximately at the junction of the rib and the transverse process The

trapezius the rhomboids and the serratus muscles are divided in a muscle-splitting or cutting type of incision The third rib is exposed for a distance of 2 inches and cleared of all soft tissues In similar fashion the third transverse process also is dissected free

The pleura is adherent to the underside of the rib and the transverse process at this area and care must be taken to avoid injuring it The finger is perhaps the best instrument to push the pleura off from the ribs and the transverse processes The rib is resected 2 inches lateral to the transverse process and removed The transverse process is then rongeured away to the body of the vertebra

A brain spoon may be used to help depress the pleura at this stage The second and third intercostal nerves are identified and a silk thread is passed around them

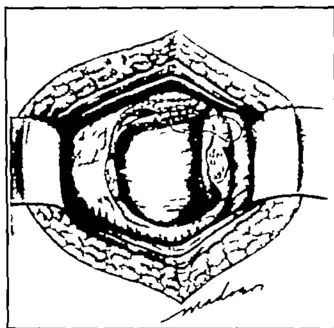


Fig 161 —Thoracic Sympathectomy The 2nd and 3rd intercostal nerves have been resected. All branches from the sympathetic chain to these nerves and to the spinal cord have been divided The sympathetic cord has been sutured into the intercostal muscles without injuring the ganglia

Tension with a nerve hook on these nerves will bring the sympathetic chain into view The chain can be held on tension with a silk thread or with a dull nerve hook while it is dissected free The chain is divided below the third dorsal ganglion The grey and white rami entering the third and the second ganglion are carefully dissected and removed by exeresis The fibers entering the spinal canal are pulled directly from it At times a drop or two of spinal fluid will be seen to escape during this procedure This method seals the fibers from regrowth in the dura

All connections from the ganglion and chain to the second and third intercostal nerves are likewise removed The chain and ganglia are dissected up to the stellate ganglion and the communicating fibers to the stellate ganglion are excised but without disturbing the ganglion The

dissected ganglia and chain are then pulled out from the thorax to be sutured into an intercostal muscle, as shown in Figure 161 In this way, all sympathetic fibers which can be visualized arising from the upper thoracic ganglia to the intercostal nerves and to the spinal canal have been divided

One end of the sympathetic chain is in the thorax and the other is outside the thorax and buried in muscle To be certain that no additional fibers are left, a 2-inch section of the second and third intercostal nerves is removed

If the pleura has not been opened, the wound is closed in layers If the pleura has been injured inadvertently, this opening is enlarged and a catheter is placed in the pleura during the closure of the wound If there is any doubt, the pleura is openly incised and the catheter left in place until the wound is closed The danger is not in the pleura being entered but in not knowing that it was entered, and a tension pneumothorax thus formed

The muscles may be closed with chromic No. 1 catgut in the line of their fibers. This closure should be extremely accurate and adequate if the pleura has been opened to prevent any air leak No permanent drain is left *in situ* The skin wound is closed with fine steel wire If the pleura has been opened, the lung is then inflated by the anesthesiologist while the pleura is sucked dry with a syringe attached to the catheter which has been left in the pleura All the air and bloody fluid are aspirated from the pleura, and the catheter is then removed A mattress suture placed at the site of the catheter is tied and the dressings are applied

(2) *Thoracic Sympathectomy with Ganglionectomy* —Many times in the previously described technic the thoracic chain inadvertently was divided or removed Anatomically it lies on the heads of the ribs and in close proximity to the transverse processes and the pleura The experiences of the author showed little variation in the results when the chain was removed Often after ineffectual sympathectomies with the technic as described in *1*, the removal of the chain was effectual In some patients, the technic described in *1* has been followed with excision of the ganglia of the 2nd, 3rd and 4th sympathetics, division of the sympathetic chain below D IV and resection of segments of the 2nd, 3rd and 4th intercostal nerves The stellate ganglion is not disturbed This procedure has worked satisfactorily in most instances, but again there is a small percentage in which the results are partially or completely unsatisfactory In this technic the 3rd and 4th ribs are resected, and in approximately 1 out of 3 patients the pleura is opened

(3) *Thoracic Sympathectomy by Open Thoracotomy* —Because of the dissatisfaction with the results of thoracic sympathectomy by any of the advocated approaches an operation was devised and performed which has been more effective This operation is performed by open thoracotomy through the pleura An intratracheal anesthesia is required for this operation The fifth rib is removed or its space spread The pleura is openly incised The sympathetic chain is readily identified on the vertebra just as it is in the thoracolumbar sympathectomy The cord is dissected up to but not including the stellate ganglion The cord is divided below the 4th or 5th ganglion The chain is then treated as in the previously described pro-

cedures with the resection of the white and gray fibers and sometimes sections of the involved somatic nerves The chain in this technic is removed The wound is closed with a catheter left in the pleura The closure must be airtight in all layers The lung is inflated and the catheter is aspirated for air and fluid If this is minimal the catheter is removed Where there

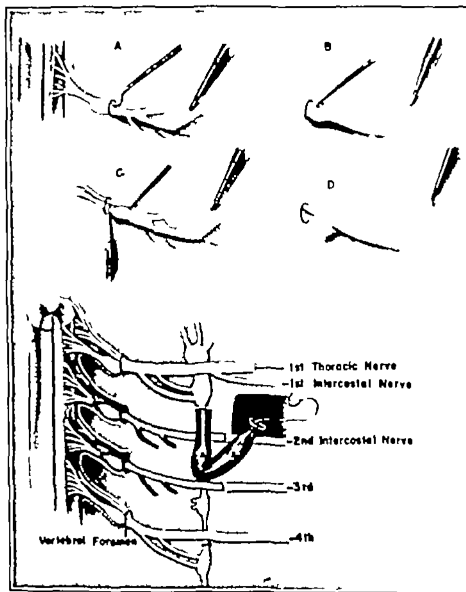



FIG 162.—Diagrammatic demonstration of the part of the sympathetic and nervous system removed in the Smithwick White thoracic sympathectomy The shaded parts of the picture show the removed segments.

Picture courtesy of Dr James White In his technic the chain is enclosed in tantalum foil and buried in the muscles

is a considerable collection of fluid or the airtight closure is under suspicion the catheter may be connected to underwater drainage for twenty four to forty-eight hours This operation appears to be better than the previous ones as identification of the chain and ganglia is technically simpler (For complete details see Direct Thoracic Sympathectomy pages 519 to 520 )

(4) *Anterior Thoracic Sympathectomy*.—A transverse incision is made directly above the clavicle just over the clavicular insertion of the sternocleidomastoid muscle. This is similar to the incision for scalenotomy. See pages 476 to 477. The sternocleidomastoid muscle is retracted medially. The avascular space is bluntly dissected. The transclavicular and transscapular branches of the thyro-cervical trunk are displaced. The scalenus anticus muscle is elevated by a ligature carrier and divided on tension. The lower cervical sympathetic chain is then located at the base of the transverse process of the last cervical vertebra and the neck of the 1st rib. Tension on this nerve brings the thoracic portion into view. The 2nd and 3rd ganglia are then divided and resected. When anatomically successful, this operation gives excellent results. Since many of the conditions for which thoracic sympathectomy is performed are benefited likewise by scalenotomy, the anterior approach, when successful, is doubly effective.

**Postoperative Care.**—Care after a thoracic sympathectomy consists of observing the patient carefully for *tension pneumothorax*. The symptoms of tension pneumothorax vary from none with mild degrees of collapse to complete apnea. The symptoms are (1) dyspnea, (2) increased pulse rate, (3) cyanosis, (4) collapse, (5) shift of the heart to the side of the pneumothorax, (6) hyper-resonance on the involved side, with distant or absent breath sounds, (7) roentgen ray evidence of pneumothorax and mediastinal contents shift, (8) when a needle is introduced into the pleura on the affected side, air under positive pressure may be removed.

Because of the danger of tension pneumothorax, it is wise to have a sterile aspirating set available at the bedside. *Vago-vagal complications* and cardiac arrest theoretically are possible. Close observation is required. Treatment has been outlined under sympathetic block and cardiac arrest. (See pages 46, and 497.)

The wound is dressed in one week. At this dressing, one-half of the sutures are removed and the other half one week later. The patient may be out of bed at once, unless there are some chest complications. These complications are reduced markedly if the patient is up. Active motion of the part should begin at once. If the operation has been performed for causalgia or some other long-continuing illness, rehabilitation should be started immediately. This is important, as many of these patients have a great fear of using the part again. Thus active motion and use of the part should not be delayed.

**Tests for Completeness of Sympathectomy.**—The degree to which the sympathetic denervation has been complete usually can be determined at operation time. This can further be checked by skin resistance changes, sweating patterns and skin temperatures determined after operation. To measure the sweating patterns two methods have been devised. (1) Chemical Method.—The basis of this test was the interaction of the skin surface moisture with a substance on the skin which has colorimetric potentialities. Originally starch and iodine were used.[54] This method gives a rough estimation but is still an indicator. The dye quinizarin (2-6-di-sulphonic acid), which is a red-brown color indicator, is now used more often.[31a] The patient must be overheated to bring out the sweating tendency, and therefore the test is not applicable immediately after opera-

tion (2) The electrical method is based upon the fact that the electrical resistance between two skin electrodes varies with the amount of activity of the sweat glands in the skin under these electrodes [10 14 77 89] Thus, one can determine quite effectively with such a skin resistance mechanism whether the sympathectomy effect is complete and the extent to which the part has been denervated (3) Skin temperature readings also show sympathectomy effect With such methods it has been determined that to denervate the upper thigh the upper lumbar and lower thoracic ganglia must be removed completely In a similar manner it is proven that to get sympathectomy effect in the toes the lower lumbar ganglia must be removed completely

## REACTIVATION OF THE SYMPATHETIC SYSTEM

The sympathetic system may become active again At the end of two years 20 per cent will show some evidence of sympathetic effect In six years the percentage of re-establishment may be as high as 50 per cent This most often is not an actual re-establishment but is due to technical failure to interrupt completely the chain In some patients branch fibers when followed will be seen to interconnect and a collateral-like re-establishment around a simple sectioning of the chain anatomically is possible

For this reason wide interruption of the sympathetics is advocated rather than mere division and excision at either end Failures are much more often due to incomplete sympathectomy than they are due to a regrowth Divulsion or exeresis gives us better results than mere division This is similar to the phrenic nerve surgery where it was found that an exeresis, instead of a division or crushing was necessary to obtain the desired permanent results.

## DIRECT THORACIC SYMPATHECTOMY

Like most surgeons our equivocal results in thoracic sympathectomy has concerned us greatly Excellent results in one patient were followed by a negative effect in the second one Often the early results were temporary Our inability to prophesy the results added to our surgical discomfort It has been recognized for a long time that surgical sympathectomy in the lower extremities is relative to the completeness of the procedure

More recently we have performed a thoracic sympathectomy similar to the technic used for the lumbar sympathetics

*Technic* —A lateral intercostal incision is made centering over the fifth interspace. The ribs are spread with the Finnochietto spreader Through this transpleural approach the lung is deflated partly and retracted forward and medially with the first assistant s hand This exposes the posterior parital pleura. The finger can palpate the sympathetic chain This pleura is incised the chain elevated from its position on the heads of the ribs It is dissected free from below D 1 to D-5 As in the lumbar area, each branch and nerve filament is dissected free and divided The ganglia are removed with the chain Care is taken to avoid injuring the intercostal vessels Sympathetic connections to the intercostal nerves are resected The wound

is closed by re-approximating the ribs, closing the muscle and fascia in three layers and being certain the wound is airtight A catheter is placed in the pleura This is aspirated as the lung is inflated at the end of the operation If there has been little or no bleeding and the closure is airtight, the tube is removed The tube can be left as underwater drainage for twenty-four hours

*Results*—The results in the patients so treated have been much better than the previous sympathectomy effects Anatomically, the number of filaments makes it appear that such a direct and anterior approach will be the only effective measure to thoroughly denervate the upper extremities.

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Chapter

28

# THE SURGICAL TREATMENT OF HYPERTENSION

*Essential Hypertension Hypertension Due to Pheochromocytoma Hyperactivity of Adrenal Glands Hypertension Due to Renal Block*

## ESSENTIAL HYPERTENSION

It is now recognized that disease of any part of the cardiovascular renal system will affect the rest of the system Thus a disease of the arteries such as arteriosclerosis often will be counterbalanced by cardiac hypertrophy Occlusion of the renal vessels will result in cardiac and hypertensive changes It is only recently that this concept of the circulatory system as a whole one has been accepted No longer are the cardiologists interested only in electrocardiographs and the word peripheral rightly has been removed in discussing vascular diseases The treatment of hypertension therefore must be considered only part of the therapy as the raised blood pressure is but a symptom of some underlying pathologic change The elevated blood pressure may require treatment because it causes such alarming symptoms The overall picture however must be kept in mind

It has been found that there is a close relationship between certain patients with hypertension and the sympathetic system Surgical denervation of sections of this sympathetic system will lower this increased pressure Hence the surgeon at times enters the therapy field

A basic problem is at once apparent since the point at which the blood pressure becomes hypertensive varies in different individuals For many years a systolic pressure from 140 to 160 mm of mercury and a diastolic pressure from 90 to 100 mm of mercury arbitrarily were considered the upper limits of normal It has been shown that there is a smooth progression upwards in pressure to the age of fifty in the male while the pressure is much more fluctuant in the female The diastolic pressure consistently but slowly rises in both sexes The influence of height weight and age are definite Recently a more moderate view on what is hypertension has been stressed [31] The fact that many people live to extensive old age even with blood pressures over 110 diastolic has been reported many times particularly in those over fifty years of age Before labeling the patient as having an essential hypertension great care should be taken to exclude those with pheochromocytoma hyperadrenalism renal blocks etc

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dition This operation produced a lowered pressure for a time, with a reduction in symptoms, but the effect was only temporary A partial adrenalectomy on both sides was attempted but the results were disappointing By 1936, he had operated on 106 patients, mostly by adrenal denervation.

These operations and his experiments on animals in Africa made Crile conclude that there was an adrenal sympathetic complex which was the basic cause for essential hypertension He then performed operations in which he resected the celiac ganglion, broke up this sympathetic complex and denervated the aorta itself

Crile was near the correct surgical interpretation of the mechanism of some forms of hypertension although his operation, as we know it today, was a postganglionic one In association with Dr W. W Babcock, we performed many adrenal denervations, but we were unimpressed with the results

The first attempt surgically to attack the hypertension problem by sympathectomy was made by Adson and Rowntree[150] in 1925 Sufficient time has elapsed for us to be certain that we can reduce the blood pressure symptoms The correct evaluation of sympathectomy in the treatment of high blood pressure must await the passage of more time

**Etiology and Classification.**—Patients with hypertension can be divided into three main groups and a sub-group

*Primary Hypertension or Essential Hypertension* —These patients have in common an elevated diastolic blood pressure The cause is unknown as indicated by the term "essential" Their courses are divergent even when the age, duration, and degree of hypertension appear equal. In general, however, the prognosis is poor

*Secondary Hypertension* —In the *secondary* type of hypertension the blood pressure rises due to obstruction to the urinary tract, obstruction of the renal artery, terminal periarteritis nodosa, terminal scleroderma, glomerular nephritis, pyelonephritis, and primary or secondary tumors of the kidneys and the pituitary gland

*Pheochromocytoma* —A third type of hypertension, now, is well recognized This is the syndrome produced by pheochromocytoma, a tumor of the medullary portion of the adrenal gland A paroxysmal or permanent hypertension results The mechanism of the syndrome is a release of epinephrine from the tumor into the blood stream

*Hyperactivity* of the adrenal glands can cause hypertension

Varying degrees of all these types of blood pressure are encountered These range from patients with minimal symptoms to the malignant type, which is followed by early death if untreated in less than two years[37]

*Primary* or essential hypertension is a disease about which we know little Ninety to ninety-five per cent of all cases of hypertension are in this group.

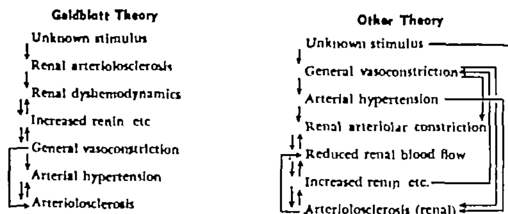
The blood pressure, in general, depends on innumerable factors, but is fundamentally controlled by (1) the cardiac output, (2) the volume and viscosity of the blood, and (3) the resistance offered to the flow of blood to the peripheral parts, particularly in the arterioles and in the capillary bed The blood pressure may be altered by the emotional state, the pos-

In essential hypertension as far as we know, neither the cardiac output nor the viscosity of the blood is altered There is however a decrease in the caliber of the peripheral vessels This is due to a change in the walls of the arterioles and/or an abnormal reaction of these arterioles to sympathetic stimulation

The part that heredity plays in this essential hypertension group has been demonstrated[4] Three per cent of children in whom neither parent has hypertension develop the condition if one of the parents has hypertension 28 per cent of the children develop hypertension If both of the parents have the condition an elevated blood pressure is found in 45 per cent of the children

In many patients with essential hypertension the sympathetic nervous system is unstable and becomes hyperactive in the presence of any unusual physical emotional or thermal stress or strain Cardiovascular hypertension disease is 5 times more common in persons who are hyperactive to

TABLE 40 —(After Wendland[41])




a standard stimulus[24] Hyper reactors are classified as those whose pressure and pulse rise when the hand is immersed in ice water

Numerous theories have been advanced in efforts to explain the mechanism of essential hypertension

Goldblatt s theory on hypertension as well as that of other investigators is summarized in Table 40

The classification of hypertension especially the essential type is difficult. The blood pressure may be elevated for a considerable period and return to normal at other times for many years before it becomes raised permanently During this period there may be few symptoms although the disease may be progressing rapidly

Hypertension developing in the younger age group has a tendency to become malignant The rise in blood pressure which comes on slowly often can be tolerated by the patient indefinitely The author observed two elderly cousins who were hypertensive but lived to the ages of seventy-seven and seventy-nine In one the blood pressure was over 240 consistently for fifteen years In the other the systolic pressure could not be read on the sphygmomanometer because of the degree of elevation Renal retentions and severe symptoms were not present in either patient.

The classification used by most investigators is based upon the retinal changes since the blood vessels, in this area, can be directly observed This has proved an effective basis for classification if its shortcomings in the borderline patient are recognized These objective signs are correlated with the symptoms and other physical findings

(1) The labile or mild hypertensive has symptoms and signs outlined in Table 41 These patients do well without therapy

(2) The second group has mild to moderate hypertension but returns to normal from time to time There is mild to moderate sclerosis of the retinal artery and few symptoms clinically of hypertension The changes, however, are established

TABLE 41 —CLASSIFICATION OF HYPERTENSION
(Modified from Wendland and Others)
Based on Retinal Changes

| *Symptoms and diagnosis* | *Character of hypertension* | *Retinal picture* |
|---|---|---|
| 1 Benign, early essential hypertension | Low grade, mild, non-progressive, labile | Normal or mild generalized narrowing of arterioles (grade 1) |

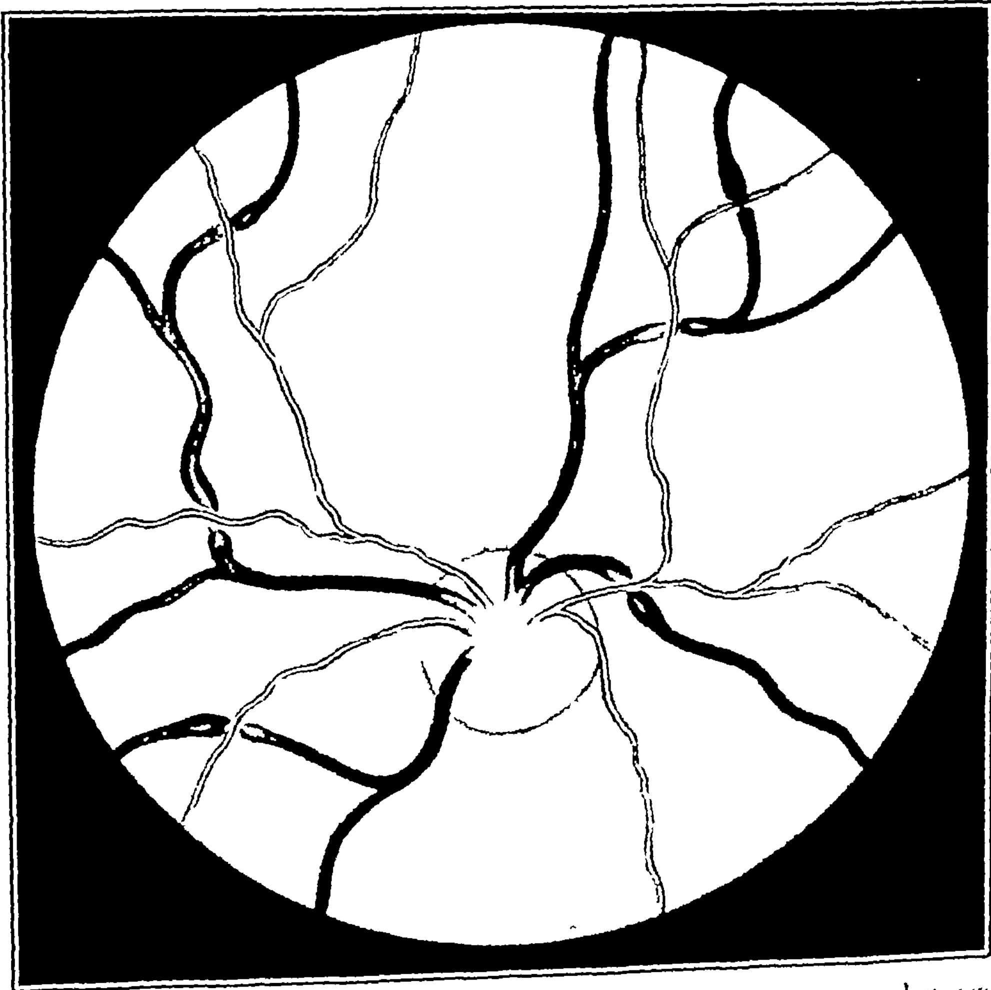


FIG. 163 —(Groups 1 and 2, Table 41 Retinal vessels show narrowing and exaggeration of the light reflex The vein is compressed (Courtesy, Sharp & Dohme Seminar)

TABLE 41 —(*Continued*)

| *Symptoms and diagnosis* | *Character of hypertension* | *Retinal picture* |
|---|---|---|
| 2. Essential hypertension benign established | Chronic hypertension of a moderate degree | Grade 1 or 2 Generalized arteriosclerosis |
| 3. Primary hypertension more severe Secondary hypertension after kidney disease mild toxemia of pregnancy | Chronic and progressive | Generalized arteriosclerosis narrowing of arterioles focal constrictions and sclerosis cotton wool patches exudates and hemorrhages |

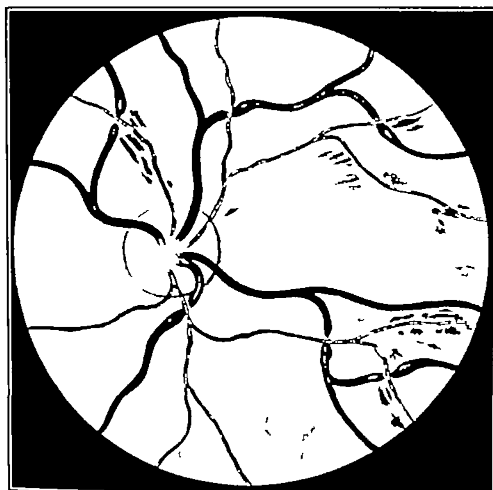


FIG. 164 —Group 3 Table 41 Primary hypertension or secondary hypertension after kidney disease Narrowing of arterioles cotton wool patches exudates and some hemorrhage (Courtesy Sharpe & Dohme Seminar )

TABLE 41 —(*Continued*)

| *Symptoms and diagnosis* | *Character of hypertension* | *Retinal picture* |
|---|---|---|
| 4. Severe essential hypertension severe hypertension following kidney disease and toxemia of pregnancy | Acute hypertension progressive unless cause is relieved often acute onset | Generalized narrowing of arterioles of all grades focal constrictions edema of retina cotton wool patches and hemorrhages |

(3) The third type is that in which there is a moderately severe hypertension, moderate sclerosis of the retinal artery, and usually clinical symptoms. In this group, there may be venous thrombosis This group borders on the malignant stage

(4) The fourth group has an acute progressive hypertension These patients may develop their hypertension acutely and will die of their disease if the cause cannot be relieved This group is the severe essential type and those with marked changes due to kidney disease or toxemia.



FIG 165 —Groups 4 and 5, Table 41 Severe, end stage Papilledema, sclerosis, cotton wool patches, hemorrhage (Courtesy, Sharpe & Dohme Seminar )

| *Symptoms and diagnosis* | *Character of hypertension* | *Retinal picture* |
| --- | --- | --- |
| 5 End stage, primary or secondary severe hypertension | Terminal, malignant | Generalized narrowing of arterioles (grade 3 and 4) Generalized arteriosclerosis, focal constriction and sclerosis, papilledema, snowbank exudate, cotton wool patches, and hemorrhages |

(e) The final or terminal stage of this disease may be primary or secondary It is a fatal disease and terminates in death early The eyeground picture is presented in Table 41 and Figures 163 164 and 165

A secondary classification of these groups can be made on the mortality statistics In the first or labile type the mortality varies from the usual death rate of that age incidence to 30 per cent A recent study of 100 patients with benign essential hypertension followed for seventeen plus years showed 71 per cent alive and 5 seriously ill [23] The second or moderate hypertensives have a mortality rate of 42 per cent in the four years after the lesion develops The third or severe hypertensive patient has a death rate of 78 per cent in the same period In the later and terminal stages the mortality rate rises to 98 per cent with the symptoms and eyeground changes as detailed in the chart These death rates are all computed on an approximate four year period

**Symptoms** —The symptoms vary with the cause type and degree of hypertension and the length of time it has been present

The clinical symptoms of hypertension are headaches dizziness fainting spells obscured vision and later blindness The peripheral edema and at times kidney and brain changes with attendant symptoms follow

There are physical findings of hypertension exudative changes in the retina cardiac effects and renal blood chemistry changes

If the hypertension is not present consistently the symptoms may be remittant The therapy which the patient has had and his response to it also affect the symptoms

*Symptoms of Pheochromocytoma* —The symptoms are detailed on page 526 but are compared here with those of essential hypertension In the hypertension which is the result of the tumor of the medullary portion of the adrenal gland a syndrome is produced in which the symptoms are similar to those that follow administration of epinephrine These are characterized by hypertension an abnormal carbohydrate metabolism and at times hypermetabolism [45] The clinical picture is that of a sudden rise in the blood pressure with increased pulse rate severe headache anxiety pallor of the face coldness and numbness of the extremities pain in the abdomen or chest at times nausea and vomiting and an abnormal perspiration The attacks may be of momentary or several hours duration They are mild usually at first but increase in severity as the tumor grows Death may follow severe attacks from a cerebral hemorrhage or pulmonary edema

The differential diagnosis must be made from the patients with essential hypertension whose blood pressures are labile and also those patients who have anxiety states or fall in the category called hyper reaction

**Pathology** —The pathologic picture varies greatly and many factors appear to influence it Among these are the age of onset the duration of the disease the height and lability of the blood pressure obesity and the resistance of the involved organs of the body The persistent elevated diastolic pressure accelerates atherosclerosis The arterioles are involved early

In the early stages spasm may be the essential lesion. In the end stage of malignant hypertension papilledema hemorrhagic retinopathy hematuria and proteinuria will be present [46] In these later stages, there

will be cardiac hypertrophy, severe sclerotic changes in the blood vessels and weight loss[39]

(a) *Arteriolar System*—Much of our understanding of the pathogenesis of hypertension is based on observations of changes occurring in the eyegrounds. These changes are considered reversible and irreversible. The reversible changes are attenuation, narrowing and spasm. The irreversible changes are arteriolar tortuosity and irregularity, the compression of the veins at the point at which the arteries cross them and an increase in the light reflex indicating more sclerosis.

(b) *Eye Ground Changes*—Hemorrhages and so-called fluffy exudates occur early in the severe type of hypertension. They may disappear under therapy. Edema is a serious sign. It is present in the cerebral phase of hypertension and also in those with malignant disease.

(c) *Cerebral Changes*—Minor cerebral vascular accidents may occur. Severe headaches may presage such accidents. Multiple retinal hemorrhages often indicate cerebral bleeding. The motor and sensory changes and faintness depend upon the area involved. Cerebral hemorrhage causes death in 15 per cent.[16] Encephalomalacia causes an equal number of deaths.[57]

(d) *Cardiac Signs*—The heart reacts to hypertension by hypertrophy. At a later date, it may fail on a compensation basis. In such event, there will be cardiac dilatation, hypertrophy and pulmonary and peripheral edema. The heart may fail due to the coronary circulation inadequacy. At such times, the coronary arteries may show sclerotic changes. There may be areas of new and old infarcts. With these changes, there will have been angina pectoris and electrocardiographic tracing variations.

(e) *Renal Changes*—The kidney is such a resourceful organ that until a disease is severe, the objective renal signs may be negligible. In the later stages, sclerotic developments in the kidney will occur with albuminuria, renal retention, and increasing depression of kidney function. Hematuria is a late stage.

**Prognosis.**—The clinical course of essential hypertension can be so divergent that it is difficult to give a prognostic picture to the individual patient. Thus, Smith *et al*, studying 2,650 hypertensive patients who came to autopsy, found only 376 or 14 per cent who could be considered truly essential hypertensives. Of these, 26 per cent died of heart disease, 20 per cent of uremia, 15 per cent of cerebral vascular accidents and 10 per cent of coronary disease, while 30 per cent died of unassociated causes. Breaking these down into the four classifications of severity, however, showed that while 60 per cent in the mild group died of incidental causes, all but 3 per cent of the severe group were lost due to their disease.[73] Keith, Wagener and Barker showed that 40 per cent of group 1, 65 per cent of group 2, 92 per cent of group 3 and 99 per cent of group 4 would be dead at the end of ten years.[26]

**Treatment.**—Many patients with hypertension respond well to medical therapy and may go on for many years living a useful life.

1. MEDICAL TREATMENT.—Some need no treatment other than observation, reassurance and organization of their habits in life on a normal level. In others, occasional periods of prolonged rest and relaxation control the

problem The variation in response particularly in the labile type makes an evaluation of any therapy difficult Spontaneous regressions do occur

No effort will be made to detail all the medical measures used in the treatment of hypertension Some of these are established some are highly controversial and in some the therapy makes the patient more uncomfortable than his disease Mention will be made of some of the conservative efforts to control the problem

*Dietary Measures* —It is recognized that certain substances when ingested may raise the blood pressure The opposite may be true Certain clinics place all their patients on a fruit diet a salt-free diet or a diet consisting of rice and others use dietary measures as only part of the therapy

*Drugs* —Certain drugs have a potent hypotensive effect One of these veratrum viride has been known for many years to be hypotensive particularly when given parenterally More recently highly purified extracts of this alkaloid which can be biologically standardized have appeared Even with such standardization assayed on the basis of the effect on dogs there is still considerable difficulty with the dosage This is due to the fact that individuals vary in their sensitivity to the drug and even react differently from time to time While far from ideal this drug appears to be one of the best pharmacologic measures available for the relief of arterial hypertension [illegible] One substance Veriloid * is given in dosages of from 10 to 18 mg per day unless hypotensive nausea develops Under 6 mg of the drug appears to have no effect Vmatesol ** is another form for intravenous use The dosage is 0.08 to 0.2 microgram per kilogram per minute Vertavis *** causes hypotension in some patients Others have reported a poor response [22] Freis who performed some of the original work with this drug cautioned against its injudicious administration [19]

Hexamethonium has reduced tension even in the malignant type of hypertension This substance has been given both parenterally and orally [11] It is the one used most often for the hypotensive anesthesia (see Chapter 3) It is given in the form of hexamethonium chloride (Methium chloride 1 6 hexane bis [trimethyl ammonium chloride]) The dosage varies with each patient and time given This drug acts by blocking parasympathetic as well as sympathetic ganglia [2] It has many side effects The drug's effect when used alone in hypertension is to make a sustained hypertension a fluctuating one A tolerance develops to the substance

Hexamethonium and Apresoline therapy (1 hydrazinophthalazine) —These two substances have been used concomitantly [42,51,56] The effect of the Apresoline alone is to inhibit or antagonize humoral pressor actions the endogenous factors implicated in the cause of some forms of hypertension [14] In combination these drugs block or abolish the neurogenic sympathetic effect and inactivate or block the circulating pressor substances [28] The result is to neutralize the psychosomatic component of hypertension

It is known that the sympathetic overactivity follows the psychosomatic part of the disease and that neurogenic action causes vasoconstriction

* Riker Laboratory Inc
** E. R. Squibb & Sons
*** Irwin Neisler & Co

which involves the renal arterioles and thus produces renal ischemia. Such renal ischemia releases pressor substances into the blood. These substances affect the body's entire vascular bed. When this arteriolar effect occurs, hypertension follows. If the hypertension persists pathologic changes in the small arteries and arterioles are permanent. These effects in the kidney continue the pressor substance release. The chain reaction appears to stop if broken for a time. It is hoped that these drugs or those similar to them may be the block the body appears to lack in those that develop malignant hypertension. The drugs cannot be given simultaneously at first. The hypotension should be achieved *gradually*. Toxic reactions and side effects have been reported. Ill effects of a sudden pressure drop are obvious.

*The Bacterial Pyrogens* have been used in the treatment of malignant hypertension. Living, non-pathogenic bacilli, dead staphylococci, streptococci, tubercle bacilli and typhoid all have been tried. Page[37,38] reports his best results with a soluble bacterial pyrogen (Pyromen).* An intravenous injection in 200 cc of saline sufficient to cause a temperature rise of 103–4° F each day, is given for as long as three to six months. It appeared from his studies that some of those doomed to renal deaths could be salvaged.[39]

*Kidney Extracts.*—The intramuscular injection of kidney extracts appears to have helped some patients with malignant hypertension.

2. SURGICAL TREATMENT.—For surgical therapy to be accepted in a condition of this type, it must be shown by percentage figures that it consistently permits those selected to live longer or at least better than they would under medical therapy. It is in the group that do not respond to medical therapy that, in selected cases, surgery may help.

It is now twenty-eight years since interruption of the sympathetics as a treatment for hypertension was first introduced by Adson,[1] working in conjunction with Rowntree.[50] Their idea was the same as ours is today: to decrease the peripheral resistance to blood flow in the vascular bed and, therefore, lower the blood pressure. Secondarily, it was hoped to stabilize the blood flow by reducing the reflex variation to the spasm factor and thus reducing the stress and strain upon the vascular bed.

Since then, Smithwick,[57] Peet,[40] Grimson,[17,18,19] Craig,[6] Weeks,[60] White,[62] Poppen,[43] and others have added to our knowledge and advances in the surgical therapy of hypertension. Weeks[60] performed the first thoracolumbar sympathectomy for hypertension in New York in 1939.

When the blood pressure is reduced surgically, no pathologic cause is corrected, although progress of the disease may be arrested. With more experience and follow-up it can be now stated that surgical treatment of hypertension in its present status should be reserved only for those patients (particularly the young) in class 4 and a few patients in class 3, provided the contraindications on page 535 do not apply.

Improvement, symptomatically, after surgery may be out of proportion to the degree of the lowering of the blood pressure. In these instances, the result may be due to the fact that the reflex spasm change is more important in that particular individual than is the reduction in the intra-arterial tension.

* Baxter Laboratories.

*Selection of Hypertensive Patients for Sympathectomy.*—Before any patient is selected for the treatment of his hypertension, the organic causes for the disease must have been ruled out. Pheochromocytoma and any blockage of the kidney or its blood supply by such lesions as tumor must be eliminated. Intravenous pyelography, air contrast *x*-ray studies, aortograms and the various tests for pheochromocytoma should be combined with the history and clinical findings to determine the accuracy of the diagnosis. Since sympathectomy is of value only in a few patients with hypertension, the problem of choosing those who will benefit by surgical treatment is important. The blood chemistry, eyeground status, electrocardiographs, heart and chest roentgenograms and cerebral evaluation first must be interpreted.

The patients should be classified as to their hypertensive status. It is more simple to rule out those who should not have surgical sympathectomy than to give indications for its use. In general, the operation is not suitable for the following:

*Contraindications.*—(1) Kidney damage with urea retention up to a level of 20 mg. per cent.

(2) Patients who have cardiac failure not responsive to medical therapy.

(3) Patients with a cerebral vascular accident within the last few months prior to operation, or in whom symptoms or eyeground changes indicate such an occurrence is expected.

(4) Patients subject to psychiatric disturbances.

(5) Patients in whom medical therapy appears effective, *i.e.*, types 1, 2 and most of 3.

(6) All patients where known causes for the hypertension exist (pheochromocytoma, renal blocks, etc.).

(7) Most patients over fifty years of age.

*Indications.*—(1) Young patients with essential hypertension (forty years of age or below) in whom the main symptoms are headache, visual disturbance and dizziness without irreversible chemistry or eyeground changes.

(2) There has been evidence that in the group 4 patients there is a significantly higher survival rate in those surgically treated than in those on a medical regimen.[illegible] Therefore, in this group, where contraindications do not exist, operation is advised.

(3) Selected patients in group 3 and, at times, group 2 may have a surgical indication.

(4) In certain patients who are hyper-reactors, the operation can prevent or retard the incidence of malignant hypertension. It should be considered for that group who show a hypersensitivity to the available tests.

It must be apparent that the rules for the selection of patients and the status of this surgical therapy at present are unsettled and subject to re-examination when a sufficient number of patients have been examined and enough time has elapsed.

There are a number of tests which should be applied to the patients for whom operation is being considered. The local conditions under which the tests are carried out should be kept as standardized as possible.

(1) *Rest Test.*—The effect of rest on the blood pressure is an important factor. In many hypertensive patients in the early stages, the blood pres-

sure level will return to normal in a few minutes or hours with rest. As the disease progresses, there comes a time when the blood pressure will not return to normal. Smithwick[57] emphasized the importance of forty-eight hours of rest before determining the basic level of the blood pressure. Perera believes that three weeks' study is necessary to evaluate the mean blood pressure in any individual.[41] The difference in the blood pressure, early, during, and at the end of the resting period, is significant.

(2) *Change of Position Test.*—Variations in the blood pressure when the patient is horizontal, sitting, and standing are important. In assuming the horizontal position, the patient normally initiates a vasoconstrictive stimulus. If such vasoconstriction did not occur when one changes his posture, there would be so much pooling of blood in the dependent position that the patient would faint from syncope. If vasoconstriction is hyperactive, as it is in the hypertensive picture, there may be an overactive response to this change of position.

(3) *Pressor Test.*[24]—The patient's hand is immersed in ice water and blood pressures are taken in the horizontal and in the upright positions. The patients are then subdivided into hyper- and hypo-reactors to this stimulus.

Most of the patients with essential hypertension are hyper-reactors to both the postural test and the cold pressor test. These tests help in selecting patients who are hyper-reactors. They are important from a prognostic standpoint. Under no circumstances should such response be used as the sole criteria for selection for operation.

(4) *Sedation Test.*—The sedation test also is used commonly to separate those patients with primary and secondary hypertension. In this test the patient is given sodium amytal, 3 grains every three hours for 3 doses. Sodium pentothal also has been used in a sedation test.

The blood pressure is taken at regular intervals for twelve hours. Those patients whose blood pressures are materially reduced under sodium amytal or sodium pentothal may be considered in the essential type and not in the secondary type of hypertension. The hypertension due to organic obstruction is little if any affected by rest.

(5) *Sympathetic Nerve Block Tests.*—Russek and his associates[51, 52] advocated the use of continuous caudal anesthesia as a test to select patients in whom sympathectomy might prove valuable. The response to the same amount of caudal anesthesia varies in patients. High levels of anesthesia have been obtained in some. We have used multiple level sympathetic blocks (paravertebral with 2 per cent novocain) as an added diagnostic aid. Enough areas to reproduce the effect of the thoracolumbar sympathectomy should be blocked and the blood pressure recorded for several hours.

The problem of selecting hypertensive patients for sympathectomy is a serious one. It is obvious, after this discussion of the various types of tests, that none of these determines the true, essential type and eliminates the secondary type of hypertension.

It thus becomes apparent that in the selection of hypertensive patients for sympathectomy one must take into [illegible] any factors. Neither the blood pressure nor the patient's r[illegible] done is sufficient for selection.

The desire of the patient for operation should be considered. In our efforts to select those who will do best, we should not fear to try the operation on certain individuals for whom the outlook is bad under any circumstances merely because we do not wish to have a fatality. A dentist who was unable to practice or to even read the daily newspaper was operated on. He resumed his practice and was active for years. Other experiences have not been as happy.

The statement of White can be modified as follows:[4] "Although it is not yet established that the results of surgery will be permanent, at least there is good reason to believe that the hands of the clock can be set back a number of years in some of the younger patients with hypertension who have not been permitted to progress to the stages of advanced degeneration of the heart, kidney, or brain."

**Surgical Sympathectomy.**—1. *Lumbar Sympathectomy.* Adson's early work with lumbar sympathectomy did not provide adequate or permanent results. This method has been discontinued.

2. *Thoracic Sympathectomy.*—The denervation of the sympathetic supply above the diaphragm was presented by Peet.[39,40] The good results he reported could not be repeated by other surgeons. This procedure in general has been discontinued because the surgical results were not sufficiently good.

**Thoracolumbar Sympathectomy.—Operative Technic.** In 1940, Smithwick[37,38] introduced the combination of the two procedures, the thoracolumbar sympathectomy for hypertension. The operative technic has varied from the original Smithwick procedure which extends from the sixth dorsal level to the second lumbar. In this technic it is important that the greater, the lesser and the least splanchnic nerves are resected. Wider and more radical resections such as those suggested by Hinton[23] and Grimson[19] have not improved the results. The operation is done under intratracheal anesthesia.

The incision is made over the most prominent lower rib, usually the ninth or tenth. *This rib is resected subperiosteally in its entire length.* The intercostal nerves above and below the resected rib should be removed. A painful neuritis may follow the failure to so resect these nerves.

The pleura is stripped away and the diaphragm manually displaced, divided, or the opening through which the chain passes dilated. The sympathetic chain is identified, lifted and dissected distally until the first and second lumbar ganglia have been freed. Then the chain is divided below L II. Sympathetic nerve connections to the celiac ganglion are divided, but *this ganglion itself is not disturbed* in order to keep the operation a preganglionic one.

The chain is then dissected upwards. The upper lumbar sympathetic chain, the greater and lesser and the least splanchnic nerves, along with the thoracic sympathetic chain are freed. The greater splanchnic nerve also must be divided at its junction with the celiac ganglion. Each of these chains is dissected cephalad at least to the sixth dorsal and frequently to the fifth or fourth dorsal ganglion. It is important to reach at least the sixth dorsal level because in most instances the greater splanchnic nerve arises from the sixth, seventh, eighth, and perhaps the ninth ganglion. The lesser and least splanchnic nerves also are resected.

The abnormal variation in the origin and distribution of these nerves makes it important to resect them to their source It is not necessary to go higher than the origin of these splanchnic nerves

When the pleura has been opened, as is usually the case, a catheter should be placed in it and left there until the wound is tightly closed At this time, the catheter is aspirated while the lung is re-expanded with the help of positive pressure from the anesthesiologist Then, the catheter may be withdrawn In any questionable case the intra-pleural tube should be connected to underwater drainage

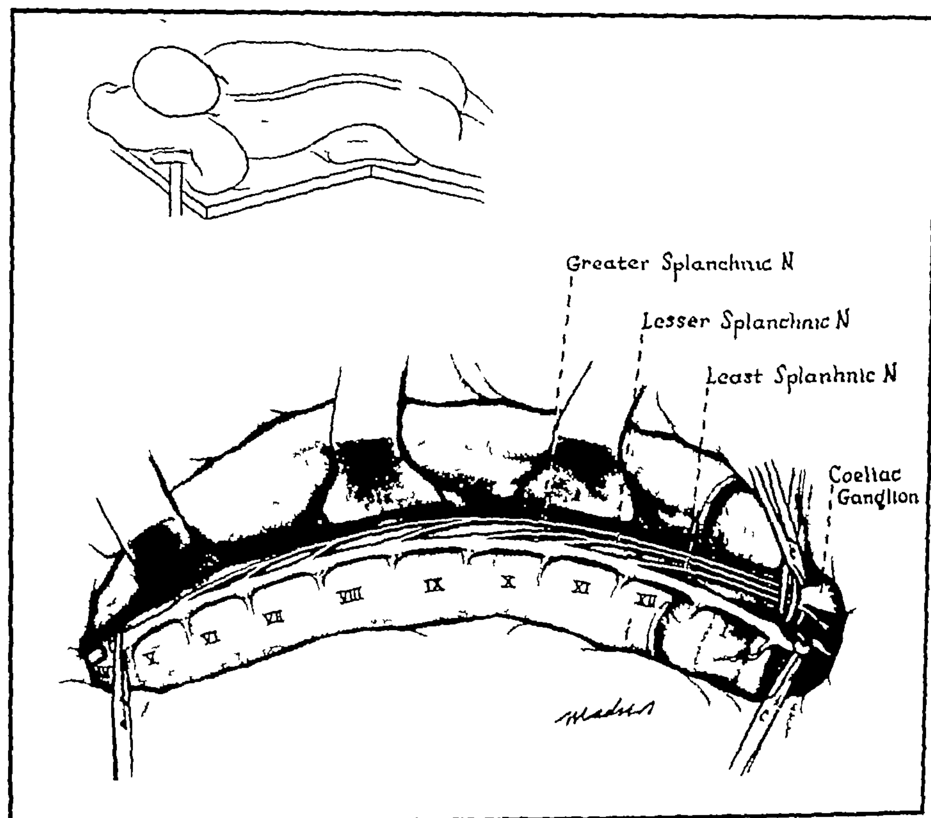



FIG 166 —Technic of thoracolumbar sympathectomy The sympathetic system is denervated from the fifth or sixth dorsal to and including the second lumbar ganglion, without disturbing the celiac ganglion Note origin of splanchnic nerves

The pleura is not sutured Closure of the wound includes (1) suturing of the diaphragm, if it had to be incised, with interrupted sutures, and (2) careful and accurate approximation of the muscles in three layers and closure of the subcutaneous tissue and skin to prevent any air leakage and pneumothorax

**Postoperative Treatment.**—After the operation, the blood pressure is watched very carefully and "Neosynephrine" is given intravenously, if necessary

As a rule, following the denervation of the first side, the blood pressure drops markedly There will be a slow rise through the following week When the second side is operated approximately two weeks later, the blood pressure may drop alarmingly for a time It may be necessary to use

"Neosynephrine" at the time and for a period thereafter to control this drop (20 mg of 1% Neosynephrine in 1000 cc saline 1 or 5% glucose). Other vasopressor substances (norepinephrine) may be used. To this measure should be added the administration of oxygen and an open, cleared airway.

After a time the blood pressure stabilizes. The diastolic pressure is an index to the results of the operation and to the prognosis.

A pneumothorax set should be kept sterile and available postoperatively at the patient's bedside. In case of any dyspnea, accelerated pulse or cyanosis, a needle should be introduced into the pleura at once and the air aspirated. If there are no untoward signs the patient may be out of bed the same or the following day. This materially reduces the complications as it does in all surgery.

One must watch for renal failure because the kidney function, as other functions of the body in these instances, may have been maintained only by the high arterial pressure. When the blood pressure drops postoperatively there may not be sufficient tension to force blood through the renal vessels and kidney substance. This same pressure problem may be a factor as it is in other parts of the body. If necessary, the circulatory system should be further stimulated for a time. Smithwick's report on 506 patients with hypertension showed 176 deaths (30 per cent) in a four- to twelve-year follow-up.[27] It must be understood, however, that there are certain disabilities and inconveniences which follow such extensive sympathetic denervation. Of these, pain is one of the most serious and it may last as long as a year. In addition the lowered blood pressure may cause dizziness. Excessive sweating occurs in the nondenervated areas. At times there is a swelling of the nasal mucous membranes.[19]

**Other Operative Technics.**—Smithwick's[26] classic thoracolumbar sympathectomy has been modified by other surgeons.

Grimson[14,15] has extended the sympathectomy to total thoracic and partial total lumbar sympathectomy, splanchnicectomy and celiac ganglionectomy. He believes he has retarded or arrested the hypertensive disease process in his patients. Others believe that including the celiac ganglion makes the operation a postganglionic one.

Freeman[9] also has performed more extensive sympathectomies. Hinton[25] continued the sympathectomy from the second lumbar to the third dorsal level, and in other cases to the stellate ganglion. Poppen[17,18] has extended the thoracolumbar sympathectomy from the third or fourth thoracic to the second lumbar level. He believes that his procedure, being more extensive, produces a more permanent and immediate lowering of the blood pressure than that achieved by Smithwick's operation.

In Poppen's technic,[18] sections of the eighth and eleventh ribs are resected and the chain is removed without dividing the diaphragm. A mortality of 1 per cent in the hospital and 6 per cent out of the hospital is not high considering the gravity of the patient's condition and the extensive surgery involved.

*Transthoracic Sympathectomy.*—The sympathectomy may be performed directly through the pleura without efforts to strip it away from the vertebrae. Since pleural stripping procedures are followed usually by laceration of the pleura anyway, this transthoracic approach is simple and

much more rapid, and the operating time is reduced The better exposure thus obtained should be followed by a better result both surgically and clinically [45]

Thorpe[59] and his co-workers reviewed 500 patients on whom there had been a sympathectomy for hypertension Their figures of 9 8 per cent excellent results, 17 per cent good results, 29 per cent fair results, 11 per cent poor results and 9 per cent unknown, are probably close to the answer of what can be expected surgically from the treatment of hypertension The 22 per cent death rate, at the time of the follow-up, showed only 3 per cent due to causes unrelated to hypertension In addition, the survival rate in groups 1, 2 and 3, at the end of three years, was no better than in those medically treated In the 4th group, however, the survival rate after surgery was statistically better

**Failure in Surgical Treatment of Hypertension.**—In the surgical treatment of hypertension there will be many failures These will be inversely proportional to the care used in the selection of the patient for operation, the elimination of other obvious causes for hypertension, the technical skill of the operation and the degree of irreversible pathology

*Poor Selection of Patient for Operation* —The mild hypertensive patient may be more of a problem after a radical sympathectomy than before as he may react to some of the denervation symptoms more severely than he did to the stimuli causing his blood pressure to rise Thus the patients must be carefully evaluated both as to the need of operation and their possible reaction to it If the lesion is too advanced failure is inevitable

*Nonessential Cause for Hypertension* —The patient with a pheochromocytoma or other hyperactivity of the adrenal gland cannot be helped by denervation of his sympathetic system He requires resection of the tumor or adrenalectomy

*Technical Completeness of Operation* —The technical completeness of the operation is an obvious prerequisite for surgical success The sympathetic system when resected must be thoroughly removed between the limits of the ganglia requiring removal if success in the selected essential hypertension group is to be expected

Reversely, some patients will respond poorly to a maximal procedure There is some evidence that the lumbar ganglia increase the effect to a greater extent than extending the thoracic part above the 7th dorsal level

*Psychic Overlay* —The psychic factors involved in hypertension have been discussed

Abnormal patients will respond equivocally due to their reaction to any deviation from the normal, *i e*, loss of sweating, paresthesia, lowered pressure, etc

Longer and more careful preoperative observation with critical selection of the patient for operation will lead to a higher percentage of successes

## HYPERTENSION DUE TO PHEOCHROMOCYTOMA

**Etiology.**—A tumor of the chromaffin system, pheochromocytoma, is a cause for hypertension It acts by secreting epinephrine and or norepinephrine into the blood stream, and 0 5 per cent of the severe hypertensions

are considered to be due to this cause. Between 500 and 800 people die annually in the United States from this condition. This hypertension occurs in any age group and equally in the two sexes.

**Symptoms.**—The main symptom is hypertension. In 3 out of every 4 patients this is a sustained hypertension and therefore indistinguishable from other forms clinically. Twenty-five per cent have intermittent hypertension. In this group there are paroxysmal blood pressure rises with palpitation, headache, sweating, tremors and in some cases convulsions and shock.

**Pathology.**—The tumor normally is small, usually single, and in 95 per cent of the cases benign. It may occur anywhere in the chromaffin system. It is most common above the kidney. Microscopically, the cells assume a brown color with bichromate stain.[41]

**Diagnosis.**—The diagnosis is made on suspicion along with other tests. Pheochromocytoma should be ruled out in each patient with hypertension. The best method of diagnosis is the provocation of an attack. This can be done by flank massage or the injection of drug. X-ray evidence of tumors is diagnostic in half the patients by intravenous pyelography and/or perirenal insufflation.

Certain pharmacologic tests exist.

(*a*) *Histamine Test.*—Rapid intravenous injection of 0.05 or 0.025 mg. of histamine base produces attacks similar to the spontaneous ones in this group. This test is positive only in those with pheochromocytoma and is accompanied by all the typical symptoms. In other patients, the blood pressure elevation is lower than that obtained by the cold pressor test.[19]

(*b*) *Tetraethylammonium Bromide.*—The injection of 100 mg. of tetraethylammonium bromide is said to raise the blood pressure and continue its elevation longer than in the patients without this lesion.[28]

(*c*) *Methacholine Chloride (Mecholyl Chloride).*—According to some authors the injection of mecholyl (25 mg.) causes a fall in blood pressure, a sharp rise in two minutes, and a return to the base level in fifteen minutes.[20] With the pressure rise the symptoms of hypertension appear if the patient has a pheochromocytoma. This does not occur in the normal or hypertensive patient due to essential hypertension unless the patient is a hyperreactor.

(*d*) *Insulin Tolerance Test.*—This test is used to determine pheochromocytoma in a patient with diabetes. If the patient has a hypertension due to a pheochromocytoma the response is the same as if epinephrine is given with insulin.[11]

(*e*) *Piperidylmethyl Benzodioxane** (933F).[14a] This drug is one of the adrenolytic substances. This test is based upon the fact that if an intravenous injection of 10 mg. per square meter of body surface was made the blood pressure would drop if it was elevated due to circulating epinephrine since this substance would act directly on the circulating adrenalin.[14] If the hypertension was due to other causes the blood pressure would remain elevated. *In a subsequent report the lesion was correctly diagnosed in 59* patients with only 3 false negative results.[14a]

* Merck & Co. Inc.

(f) *Dibenamine Hydrochloride (Dibenzyl-beta-chloroetyl amine hydrochloride)* —This substance has been used to diagnose these tumors because of its adrenolytic effect Because of the patients' reaction, its use is limited

(g) *Regitine* *—This substance can be injected intramuscularly (5 mg) and will produce a lowered blood pressure in patients with pheochromocytoma Piperoxan also is a test for circulating epinephrine in the hypertensive patient with pheochromocytoma These tests are useful and can be utilized as an adjunct in the diagnosis Uremia is a contraindication to their use as false results can be obtained [37]

**Surgical Treatment.**—The surgical treatment is excision of the tumor The surgical treatment must include an exploration of the abdominal chromaffin system as well as the adrenal areas Laparotomy incision is the one of choice During the operation a hypertensive crisis should be prevented by careful handling of tissues, the inhalation of amyl nitrite and intravenous injection of Hyperoxan or Regitine Postoperatively, the patient must be observed with blood pressure determinations at five minute intervals for forty-eight hours The preoperative administration of adrenal cortical extract or ACTH may prevent the postoperative shock General anesthesia instead of spinal is advised During operation the administration of norepinephrine may prevent the hypotensive crisis This substance may be used postoperatively and adrenal cortical extract or cortisone may be necessary

The possibility of an abnormal position of such tumors is emphasized by the resection of a pheochromocytoma from the thorax by Meier [30] Twelve per cent of such tumors have been found to be outside the adrenal area [5 13 16 21 22 27 33 47 58]

## TREATMENT OF HYPERTENSION BY ADRENALECTOMY

Following the early work of Crile,[7] resection of parts or all of the adrenals has been utilized in the treatment of hypertension It is evident that hypertension cannot exist in the absence of the adrenal cortex The operation, therefore, is somewhat on an empirical basis [57] Bilateral total adrenalectomy has been performed on 12 patients by Merrill and his co-workers [31] Smithwick suggests that subtotal adrenalectomy be performed in some of the patients who do not respond to splanchnicectomy [57] More recently the removal of 85 to 95 per cent of the adrenal gland has been reported with apparently beneficial results It appears that this operation may be of help when used in conjunction with sympathetic denervation [63] Replacement therapy is required This can be supplied by cortisone and ACTH The results of the adrenalectomy have been uncertain In some, the headache, palpitation and dyspnea have decreased In others, the heart and liver size have been reduced The blood pressure has been lowered in about half the cases There has been some difficulty in the maintenance of sodium chloride The amount of salt to maintain body economy and yet not lead to excess accumulation of salt and water is a difficult point to balance The technical points in adrenalectomy are detailed in general surgical texts

* Ciba Pharmaceutical Products, Inc

## HYPERTENSION DUE TO RENAL BLOCKAGE

Hypertension can result from any block in the renal system. A tumor of the kidney or external pressure on the kidney or its blood supply will raise the blood pressure. The symptoms are those of unrelieved hypertension combined with the symptoms of the cause. The treatment requires removal of the cause. If the condition has persisted for some time, irreversible changes may have occurred.

*Conclusion.*—In conclusion, it is fair to say that surgically we can help a limited number of patients with essential hypertension. This help may be to extend life to a few and improve the symptoms and living status of others. Surgeons can cure hypertension due to pheochromocytoma. At a later date, with continued research, more benefit may be derived operatively than can be promised at this stage.

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# Chapter 29

## ARTERITIS

**Definition.**—Arteritis is an inflammatory lesion involving one or more arteries characterized by exudates, necrosis and, at times, death of the tissue supplied by the artery. These lesions are discussed under the headings *Periarteritis Nodosa* (Polyarteritis, Essential Arteritis or Essential Polyarteritis, Kussmaul-Maier Disease), *Temporal Arteritis* (Cranial Arteritis), *Panniculitis* (Weber-Christian Syndrome) and *Non-Specific Arteritis*.

### PERIARTERITIS NODOSA (POLYARTERITIS, ESSENTIAL ARTERITIS OR ESSENTIAL POLYARTERITIS, KUSSMAUL-MAIER DISEASE)

**Etiology.**—Periarteritis nodosa is of unknown etiology. It occurs most often in females between the ages of thirty and fifty. Periarteritis nodosa was first described in 1866 by Kussmaul and Maier,[10] although Rokitansky[14] mentioned it as early as 1852. A thorough description of the disease occurs in medical textbooks.[1 14 15] It resembles an inflammation of the artery, but no specific organism has been isolated. Vascular allergy has been suggested by Harkavy,[6] causing the vessels to develop a reaction when sensitized to some agent. Rich[11 12] thought that foreign protein precipitated the lesions and produced similar changes in rabbits. The association of the lesions with rheumatic fever and asthma has been noted. It appears that there are at least two forms. In a local one, the lesions affect the superficial structures, remit for long periods of time, and may be cured or at least arrested. In a second form, the vital structures such as the bronchi, myocardium, serous membranes, the blood forming and nervous systems are affected. Dickson[4] distinguished periarteritis from polyarteritis acute nodosa. The former affects chiefly the outer vessel coat. The latter has small localized nodules on small and medium sized arteries. These result from inflammatory, destructive and proliferative changes.

**Symptoms.**—The symptoms of periarteritis nodosa are general and local, the latter depending upon the site of the lesions. The most common symptoms are pain and malaise. Associated with this pain is a marked degree of fever, with weakness similar to that seen in leukemia. There is local inflammation at the site of the lesions, and frequently there is edema distally. Muscular pains particularly are common. The course, in the

generalized disease is downhill and the patient usually dies of a cardiorenal failure There may be acute renal symptoms. Symptoms may develop in the gastrointestinal tract or lung

**Pathology** —The involved artery shows necrosis of the media infiltration of the adventitial tissue with polymorphonuclear leukocytes and some degree of destruction of the intima Most of the obstruction is in the media with encroachment of the lumen When this extends to the intima occlusion of the artery occurs with secondary avascular changes in the part supplied by the artery Hypertrophy of the endothelium may close the lumen or partially occlude it A suppurative periarteritis may be seen

If the lesion regresses the inflammation subsides and in the end-stage the vessel is entirely replaced by scar The surrounding tissue early shows the pathologic picture of inflammation and later necrosis or the scar if the process resolves Collateral vessels will dilate around the affected one and take over its functions (See Fig 167 ) Aneurysm may develop



Fig 167 —Typical pathologic section of periarteritis nodosa.

**Diagnosis** —Only 10 per cent of the cases of periarteritis nodosa are diagnosed during the patient s life The diagnosis is made on the symptoms and a suspicion of the disease on the general course and confirmed by biopsy There is a relative eosinophilia in many cases Relapses and apparent cures occur

**Treatment.**—There is no accepted treatment for periarteritis nodosa Surgically the lesions are of interest because of the value of biopsy in diagnosing suspicious lesions Treatment in general is of the changes occurring in the affected part *i e* kidney Antibiotics have been used

In 4 patients we observed remissions or cures were effected at a time when the prognosis appeared to be extremely poor These patients were treated by huge doses of penicillin and small repeated blood transfusions The small blood transfusions apparently give the patient some added lift or supply some factor of a defensive nature not already present in the patient s blood This therapy resulted in recovery in these rather hopeless

patients. While not specific, this method seems to have a logical basis and is suggested for other patients One of these patients is my mother

Surgical excision of a local lesion might be attempted if it does not occur at a site necessary for life Drainage and excision are necessary in the suppurative periarteritis patient Cortisone and ACTH have caused relapses and amelioration of the symptoms

## TEMPORAL ARTERITIS (CRANIAL ARTERITIS)

**Etiology.**—Temporal arteritis is a rare condition probably due to inflammation of the temporal artery It was first described in 1890 by Hutchinson[8] and emphasized in 1932 by Horton, Magath and Brown [8] Its asso-



Fig 168 —Typical pathologic section of temporal arteritis

ciation with arteriosclerosis has been recognized, but this may be coincidental inasmuch as all cases reported up to the year of 1946 were over fifty-five years of age A tight hat has been suggested as a cause of the lesion Its cause actually is unknown

**Symptoms** —Headache is the main symptom of temporal arteritis It may be generalized, but eventually it centers in the frontal or temporal areas This headache is sufficient to keep the patient awake at night Usually, the condition occurs bilaterally There may be fever and night sweats

Cerebral symptoms may develop varying from mild vertigo to disorientation and delirium There may be some diplopia and visual diminution Blindness has occurred With active inflammation of the temporal artery the pulsation in the vessel stops The area becomes red and extremely tender.

**Pathology**.—There is usually nodular induration in the artery. Grossly a small segment of the entire artery may be involved. Histologically there are localized patches of necrosis in the media. There are large collections of lymphocytes with periarteritis and mesarteritis. The lymphocytes and fibroblasts collect around the blood supply to the vessel itself. Thrombosis occurs. Granulation tissue replaces the media. The intima becomes markedly thickened. Unlike the local nature of the inflammation in periarteritis nodosa, that of temporal arteritis extends along the artery.

**Treatment**.—Surgical excision of the inflamed temporal artery has been effective in some patients. If the condition is more generalized, excision only establishes the diagnosis. Small repeated blood transfusions and penicillin or other antibiotics as described under periarteritis nodosa are suggested as a treatment of temporal arteritis.

Local procaine hydrochloride injections have been used by Roberts and Askey.[13] This suggests a sympathetic vasospasm as the cause of temporal arteritis.

Vitamins $B_1$, C and E should be given empirically.

It is to be emphasized that marked relief has followed surgical excision of the involved artery.

Cortisone and ACTH have helped some patients.

## PANNICULITIS (WEBER-CHRISTIAN SYNDROME)

This lesion, while quite rare as reported in the literature, has been seen sufficiently often in the recent years to consider it of more common incidence than reported. The disease is of unknown origin but follows a fairly definite pattern.

**Symptoms**.—*General*.—The symptoms simulate periarteritis. There is generalized malaise, joint pains, mild fever and, at times, signs similar to an upper respiratory infection. There is a leukopenia.

*Local*.—Subcutaneous nodules develop. The skin overlying the nodule becomes red. These nodules appear as subcutaneous tender areas not unlike an incipient furuncle. They have been incised as such. Over half of them appear on the lower extremity, with the arm and trunk next most affected. The face rarely is involved. The lesions occasionally break and drain a sterile, yellowish fluid with fat globules.

**Pathology**.—Christian[3] described the histology as cellular infiltration of the panniculus adiposus. The type of cell seems to vary from lymphocytes to polymorphonuclear leukocytes and phagocytic cells for the fat. Edema, infiltration and necrosis are common. In most instances there are some changes in the blood vessels. In a few there is periarteritis, in some an endarteritis. With these changes the part may become more susceptible to mild trauma. Thrombosis and ischemia may explain the tissue breakdown and fat necrosis. The infiltration is phagocytic and chronic inflammatory cells follow.

**Treatment**.—No effective treatment has been evolved. If excision is performed for diagnosis, it should be complete. The healing may be delayed and ulcers have been formed. Antibiotics, replacement vitamin

therapy, warm packs and small blood transfusions may be indicated The effect of the anticoagulant drugs on these lesions is questionable.

In localized lesions, wide excision and sliding grafts have been effective In others, excision and secondary grafts seem effective The adrenal cortex substance may help

## NONSPECIFIC ARTERITIS

In addition to the already described periarteritis and specific arteritis of such vessels as the temporal artery, a nonspecific arteritis may occur in various parts of the body

**Etiology.**—Most likely, arteritis occurs in many diseases Recovery with recanalization or adequate collateral vessels masks this lesion It may occur with syphilis, a septic organism, filterable virus, toxemia or on an anaphylactic basis There are no specific causes

**Pathology.**—With this lesion there are primary changes in the vessel wall There is swelling, necrosis, and there may be destruction of the media and adventitia, particularly in the elastic parts Polymorphonuclear leukocytes and eosinophiles infiltrate The medial coat may become necrotic and aneurysms have been found. As the area heals, the leukocytes decrease and granulations become predominant These granulations are changed into scar If the condition extends, the part supplied by the artery may die The pathology then varies with the site of the arteritis.

**Symptoms.**—The symptoms are not unlike that of arteritis elsewhere They depend upon the extent of the lesion and the part of the body involved In general, they are inflammatory They are frequently called "neuritis," "undiagnosed fever," or even "cardiac disease" There is usually fever, weakness, pain, loss of weight and local signs, depending on the part involved

**Treatment.**—No specific treatment can be detailed Antibiotic and anticoagulant therapy should be used In questionable cases biopsy may diagnose the condition and be therapeutically helpful In localized lesions, excision may be curative The adrenal cortex substances may be tried

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# SECTION V

## The Venous System

## Chapter 30

# VARICOSE VEINS

*Anatomy, Pathogenesis and Surgical Treatment*

## ANATOMY

THE venous system of the body consists of two distinct types of vessels—the pulmonary and the systemic systems These vessels have as their function to carry the blood from the capillaries in the different parts of the body to the heart The portal venous system is a part of the systemic vein system but its action is confined to the abdominal cavity It transports blood from the digestive parts of the body and from the spleen to the liver From this latter organ the blood is returned to the general system by way of the hepatic veins which empty into the inferior vena cava This latter system and its relation to the vascular surgical problems is considered in the chapter on esophageal varices and portal hypertension See pages 752 to 754 The pulmonary venous system returns blood from the lungs to the left heart It is unlike all other venous channels in that it contains oxygenated blood It has been considered surgically under the chapter on treatment of cardiac lesions (See page 105) In general the course of the blood through the body begins in the left ventricle and this circuit is called the systemic or greater circulation until such time as the blood reaches the right auricle Its passage from the right auricle through the right ventricle to the lungs and then to the left side of the heart (left auricle) may be called the pulmonary or lesser circulation Thus arteries are the vessels which convey blood from the heart and veins return the blood to the heart During fetal life the blood varies in its course and arteries and veins may transport both arterial and venous blood at different times Normally such duplicate channels cease to exist prior to birth

While both the arterial and venous systems are vital parts of the general circulation they are actually two distinct and separate systems being connected directly only in the capillary bed

The venous diseases of surgical interest in this chapter are those of the systemic system which carries the blood from the body back to the right atrium of the heart.

Anatomically, the systemic venous system is divided into the superficial and deep systems and the venous sinuses. The venous sinuses are found only in the interior of the skull, and are of no concern at the moment. The superficial veins lie between the layers of the superficial fascia beneath the skin. They drain mainly this area and the skin. They connect with the deep veins by perforating branches through the deep fascia. Deep veins accompany the arteries and are usually in the same sheaths with these vessels. The veins accompany arteries, except in the liver, the skull, and the vertebral column. The larger arteries have one vein accompanying them. In the smaller arteries, such as the radial, ulnar, or tibial, the veins occur in pairs, the venæ comitantes, one lying on either side of the artery.

The veins of the upper extremity, head, neck, and thorax end in the superior vena cava. The veins in the lower extremity, the abdomen, and pelvis terminate in the inferior vena cava. Both cavæ terminate in the right atrium.

The veins are provided with valves. There are many more valves in the deep than in the superficial veins. In the veins of the lower extremity there are many more valves than in the veins of the upper extremity. Pathologically, the veins of the lower extremity are involved in a large percentage of venous diseases.

**Superficial Veins of Lower Extremity.**—The superficial veins of the lower extremity are the greater and lesser saphenous veins.

The *great saphenous vein* is the longest vein in the body. It begins as a marginal vein in the dorsum of the foot and ends in the femoral vein approximately at the inguinal ligament, where it joins the femoral vein after passing through the fossa ovalis. In its course, the great saphenous vein is medially placed, ascending in front of the internal malleolus and behind the condyles of the tibia and femur. It receives tributaries by free anastomosis with the lesser saphenous veins and the anterior and posterior tibial veins. Near the fossa ovalis, it has multiple branches which are of importance.

The *lesser saphenous vein* begins as a continuation of the lateral marginal vein of the foot behind the external malleolus. It ascends as the lateral marginal vein to the tendo achillis and then runs up the middle of the back of the leg to perforate the deep fascia, usually at the junction of the middle and upper third of the leg. It lies on the fascia between the two heads of the gastrocnemius muscle to drain into the popliteal vein. Occasionally, this lesser saphenous vein perforates the deep fascia just above the insertion of the tendo achillis. Over the tendo achillis the lesser saphenous vein is joined by a branch running from the anterior part of the ankle and foot. Although not described in anatomy books, this branch is demonstrated routinely with the vein stripper in varicose vein operations.

**Deep Veins of Lower Extremity.**—The *deep veins* of the legs accompany the corresponding arteries. The deep veins arise as digital veins on the plantar and dorsal surfaces which intercommunicate. These plantar veins become the metatarsal veins which unite by perforating veins with the veins on the dorsal surface to form the deep [illegible] venous arch which corresponds to the arterial arch. These p[illegible] [illegible]s run medially and laterally, communicate with the saphenou[illegible] [illegible]l then unite behind

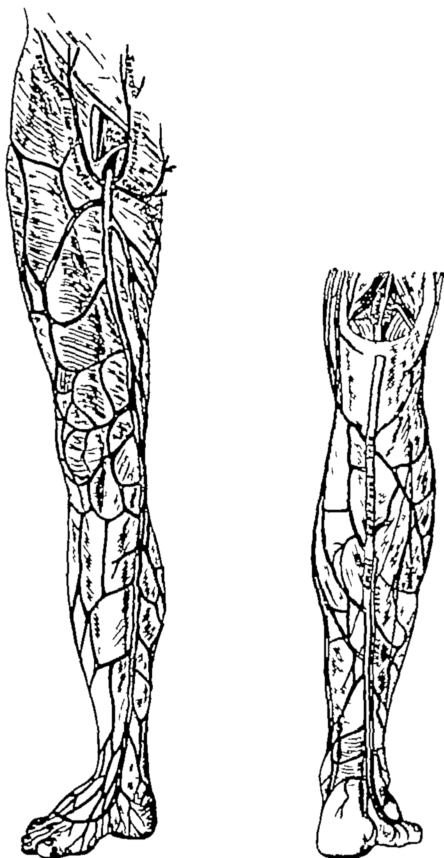


FIG 160 —Great saphenous vein and the small saphenous vein (Gray's Anatomy

the internal malleolus to form the posterior tibial vein This vein follows the course of the same artery and is joined by the peroneal veins The anterior tibial vein is a continuation of the venæ comitantes of the dorsalis pedis artery. The anterior and posterior tibial veins join to form the popliteal vein at the lower border of the popliteus muscle The popliteal vein then ascends to pass through the adductor magnus muscle, at which point it becomes the femoral vein

The femoral vein accompanies its artery It is lateral to the artery in the lower part of the leg, and directly behind it in the middle of the leg At the inguinal ligament, it is behind and slightly medial to the artery More recent study indicates the femoral vein has an average of three valves, rarely none, and uncommonly as many as six Ninety per cent of the femoral veins have a valve distal to the profunda These valves are bicuspid with an anterior and posterior cusp [2]

The femoral profunda vein joins the femoral vein approximately 1½ inches below the inguinal ligament The femoral profunda vein is made up of perforating branches similar to those of the profunda artery It communicates with the popliteal vein below and with the inferior gluteal vein above The medial and lateral femoral circumflex veins also join with profunda veins The profunda and femoral veins form the common femoral vein

The common femoral vein has a valve at its upper end, usually within 1 cm of the inguinal ligament [2] It then becomes the external iliac vein behind the inguinal ligament Approximately 1 in 4 iliac veins has a valve [2] This external iliac vein is united with the hypogastric vein at the sacroiliac articulation to form the common iliac vein On the left side, this iliac vein is on the medial side of the artery On the right side, however, it lies medial to the artery in the lower part, but in the pelvis, it passes behind the artery

Valves in the common iliac vein occur but are rare. These common iliac veins join at the level of the fifth lumbar vertebræ on the right side to form the inferior vena cava Valves are not found in the vena cava [2] In their course, these veins have received branches from the gluteal, the pudendal, the obturator, the hemorrhoidal, the pelvic, and the sacral veins The pelvic veins are important when they become involved in thrombotic or infectious processes The vesical plexus, draining the bladder and the prostate, empty into the hypogastric veins The veins of the penis are two in number. The superficial vein drains the prepuce and skin and enters the superficial external pudendal vein, a branch of the great saphenous vein The deep vein receives blood from the glans penis and corpora cavernosa of the penis and becomes the chief tributary of the pudendal plexus and enters the hypogastric vein In the female the uterine plexus is emptied by a pair of uterine veins which drain into either hypogastric vein The vaginal plexus opens into either hypogastric vein by a vaginal vein The spermatic veins arise from the back of the testis, receive branches from the epididymis and then unite to form the convoluted pampiniform plexus which is the mass in the spermatic cord. These vessels enter the abdominal wall through the inguinal canal and ascend on the psoas muscle on either side of the internal spermatic artery They unite to enter the inferior vena cava on the right side at an acute angle. On the left side, they enter the left renal

vein at a right angle This angular entrance is believed to be the cause for the left scrotum being lower than the right and the increased incidence of varicocele on the left These vessels have valves The ovarian veins similar to the spermatic veins begin as a plexus in the broad ligament near the ovary and tube On the right side they end directly in the inferior vena cava On the left side the drainage is to the left renal vein This anatomical variation is of importance in pelvic thrombosis and its treatment.

The anatomy of the venous system plays a part in the development of the pathologic changes and diseases to which it is susceptible

Diseases of the venous system are responsible for a high percentage of hospital admissions. In large general hospitals 5 per cent of hospital admissions are in this category In hospitals which maintain a vascular clinic this figure becomes 10 per cent




FIG 170 —Popliteal Vein. Muscle perforating branches sites of possible thrombosis when pressure is exerted on the calf (Gray's Anatomy )

The diseases of the venous system may be congenital or developmental in nature

A large percentage of venous diseases are due to varicose veins

## VARICOSE VEINS

**Etiology** —In simple varicose veins the vessel walls dilate In most instances (85 per cent)[21] there are incompetent valves The causes for such dilatations and valve failure may be multiple but can be divided into fundamental and precipitating causes

1 FUNDAMENTAL CAUSES —A *Inheritance* – In 70 per cent of the patients a congenital weakness of the venous system is derived from the parents. In such individuals the wall and valve structure is defective The stress of life causes such patients with fundamental defects to develop varicosities

B *Age* —The vein wall and valves lose tone with increasing age In our study of 436 patients who were over forty years of age and actively working 42 per cent of the males and over 70 per cent of the females had pathologic enlarged veins[22]

C *Sex* —The female sex presents not only a higher percentage of the patients with varicose veins but also those with the more advanced pathology The reasons for such sex difference have been argued for years The greater angulation of the vascular structures by the broad pelvis in the female may play a part, as also may the pelvic organs The congestion of menstruation has been considered a possible cause. The poor posture of the female in general and the high heels and faulty arch supports likely contribute

D. *Assumption of the Upright Position* —The fact that man walks on his "hind legs" has been blamed for many of our ills In venous diseases it is not unlikely that it plays a part The venous return from the lower legs must ascend about 4½ feet against gravity With arch defects there is alteration in the muscle support, particularly in the adductor group, through which the deep veins return Varicose veins are not seen in such four-legged animals as the horse and dog, although these two are subjected to many of our own living conditions This position factor alone is not sufficient to cause the varicosities or the incidence would be greater than 10 per cent

2 PRECIPITATING CAUSES —A *Physical or Chemical Constrictions* —Garters, girdles, elastic bands and supports, tourniquets, braces, casts and bandages are all possible aggravants It is believed that the periodic use of vasoconstricting drugs may play a part Thus, adrenalin, ergot and nicotine are likely offenders

B *Posture* —Poor posture of a congenital or acquired nature can bring out latent varicosities The pathogenesis may be similar to that caused by the scalenus muscles in the neck Torsion and back pressure on the venous structures may follow defective positions

C *Obesity* —Like nearly all other ills, the overweight patient develops varicose veins more often and in greater severity than the thin individual.

D *Pregnancy* —In the individual who is subject to varicosities, pregnancy often will precipitate them Each subsequent child-bearing and labor further aggravates the situation The number of women who have children without any pathologic change in their veins, however, demonstrates that pregnancy alone is not the cause

E *Occupation* —Like pregnancy no particular occupation causes varicosities. The underlying weakness must be present This needs emphasis at this time in our changing social order when any defect or sickness may be considered compensible It may be possible, however, that standing in one position may inaugurate the symptoms earlier and to a greater degree than in those who walk or sit The elevator operator, the dentist, the traffic policemen and the barber more often present symptoms than the mailman, soldier or delivery man. Again the number of persons who stand without developing varicosities proves that standing, alone, does not cause the lesion

F *Trauma* A direct injury may weaken the vein wall or valve An indirect injury which causes an increased venous pressure by strain may bring out the inherent weakness Most often an injury causes a clot A subcutaneous hematoma may be followed by varicosities The varicosities following a blood clot are discussed in the chapter on thrombosis, p 561.

G *An Increase in the Intravenous Pressure* —This may be due to external pressure or strain The venous pressure varies with the position of the body and also with exercise The pressure in the superficial veins of the leg is equal approximately to that of a column of fluid of the same height (100 mm of mercury) When an individual begins to walk there is an increase in pressure at first The pressure then drops to 70 mm After

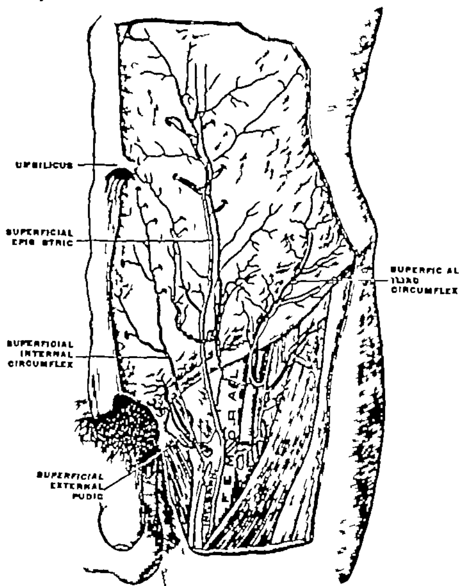



FIG 171 —Anatomy of vascular system in the groin (Gray's Anatomy ) Note that the femoral artery is shown lateral to the vein when actually it is directly over it The saphenous vein and its branches may increase in size to 3 times that shown in the picture

exercise the pressure may be as low as 50 mm and this gradually returns to normal A variation in position exercise or disease status can alter this venous pressure markedly Thus a mild venous valve failure can so vary the venous pressure as to produce further valve failure [illegible]

*In patients with varicose veins and secondary edema or ulceration* the venous pressure is not reduced by exercise and may be increased As there is no tendency to empty the veins in these individuals by walking the mean venous pressure is elevated rather than reduced The pressure gradient

from arteries to veins is reduced with increased intracapillary pressure, thus forming more edema

The varicosities which develop secondary to cardiac, hepatic, renal, portal or pulmonary disease are not simple varicose veins. Their etiology is apparent The vein enlargements which follow blockage of the normal drainage by injury, scar, burn, new growth or other compressions are not simple varicose veins Their cause also is obvious

In addition to simple varicose veins, there are two other types of enlarged veins which may confuse the picture These are discussed under pathogenesis on pages 561 to 562 They are the following (1) Varicosities which occur following thrombitis (thrombophlebitis) of postpartum, postoperative, traumatic, infectious or chemical origin See the chapter on Thrombitis, p 616, for details (2) Arterial varices in which the veins enlarge due to small artery connections The subject is discussed in detail in the chapter on Arterial Varices, p 589.

## PATHOGENESIS

**Simple Varicose Veins.**—To understand the development, symptoms, and complications of varicose veins, a knowledge of the anatomy and pathology of the condition is essential (See Anatomy)

The venous return from the lower extremities is dependent upon the femoral and saphenous system The deep femoral system, well supported by heavy muscles, is rarely the site of varicosities The greater and lesser saphenous systems subcutaneously placed, unsupported by muscles, and subject to pressure and trauma, are the common sites of the pathologic varicose veins The great saphenous vein drains the medial aspect of the leg and empties into the femoral vein at the saphenous-femoral junction

A venous backflow from the femoral into the saphenous system, at this main junction, normally is prevented by the saphenous-femoral valve This bicuspid or rarely tricuspid valve becomes incompetent in 85 per cent of those with varicose veins[2] With this valve incompetency, the saphenous system is unable to empty itself The femoral vein blood tends to dam back into the saphenous tree, causing dilatation, further valve damage, and stagnation of the blood in the saphenous vein and its branches

The great saphenous vein also communicates with the femoral vein at many other points besides the saphenous-femoral junction—the so-called communicating branch veins The valves guarding these communicating branches also may become incompetent and permit femoral vein blood to backflow to the great saphenous vein at these points.

The lesser saphenous vein, which drains the superficial part of the posterior part of the calf and empties into the popliteal vein under the deep fascia between the heads of the gastrocnemius muscle behind the knee, also is frequently involved in varicose vein development

The superficial skin and subcutaneous tissues, drained by the saphenous veins, thus become congested and edematous, and the tissues are constantly bathed in stagnant, deoxygenated blood Secondary skin changes occur with edema and later dermatitis A brown, red, or even black discoloration appears caused by a deposit of hemosiderin pigment

This skin change is complicated by an invasion by a fungus infection With scarring, eventual ulceration occurs Fungi in one form or another

(Trichophyton) are universal contaminants They are present on all public baths and beaches In patients with venous congestion trichophytosis is more common as the dampness of the tissues encourages the growth of the fungus Some form of Trichophyton can be cultured from nearly all ulcers Even in those from whom a fungus cannot be isolated mild fungicides improve the local picture of the ulcer a therapeutic test of their presence Other skin lesions of an eczematoid type may appear A slight trauma causing a skin break will not heal The local defenses of the area are poor and secondary invasion of the staphylococcus or streptococcus is frequent

**Postthrombotic Varicosities** —Not to be confused with simple varicose veins are enlarged veins that follow an inflammation or so-called thrombophlebitis or thrombitis These enlarged veins are an entirely separate and different entity They are discussed in detail under pathologic venous clotting Such enlarged veins result from the inflamed veins that follow typhoid fever diphtheria influenza pneumonia operations trauma, and childbirth

In thrombitis which follows an operation an obstetrical delivery, some infectious disease or an injury the intimal wall of the veins becomes traumatized This trauma may be due to the pressure of the walls of the veins against each other as occurs to the veins of the calf in bedrest Concomitantly there is some alteration in the intravascular clotting factor and stasis, producing a clot in the vein This clot may be accompanied by inflammation (thrombitis) or be without inflammation (thrombosis) When this condition occurs collateral veins develop around the clot in an effort to aid the venous return

This picture is different in its origin pathologic basis, and surgical treatment from that of the simple varicose veins *This differentiation must be understood* and must be kept in mind whenever a patient with dilated veins is encountered Serious and even fatal complications have followed when the postthrombotic varicosities have been treated as if they were simple varicose veins

The veins thus developed are due to the need for collateral venous return, with the closure of a main vein With recanalization of a main vein the large collateral veins remain as venous by passes The difficulty with which this thrombitis type of varicose veins responds to treatment places them in a separate category [17, 30] See page 620

**Arterial Varices** —For many years, surgeons have been aware that abnormally large varicosities develop in certain individuals which do not respond to the usual surgical measures In these individuals, enormous dilatations of varicosities occur sometimes over a very short period of time The pathologic picture advances so rapidly and is so extensive that it cannot be explained on a valve failure basis In patients who have recurrent varicosities immediately after operation the same picture exists

For a time these recurrences were considered due to technical failures of the operating surgeon Since many of these operations were performed by skilled surgeons who had the fundamental principles of vein therapy thoroughly in mind it was necessary to look elsewhere for the cause of the failure

In operating on such patients, the veins when opened had an arterial pulsation, synchronous with the heart beat There were direct arterial connections, either from the pudendal branch of the femoral artery or, at times, from the femoral artery directly opening into the superficial system of the saphenous vein Such arteriovenous connections most often occur in the veins which are seen on the lateral aspect of the thigh and calf and in the popliteal space

These findings have been confirmed so many times that enormous dilatations on the lateral or posterior aspect of the leg are suspected at once of being arteriovenous in origin Many small arteries open into a superficial vein, pump rapidly into it and dilate it enormously Wright[39] too has noted this entity

Clinically, in these patients, we see arterial blood actually entering the venous tree. This factor should be considered (a) in each instance in which there is sudden and enormous dilatation of the venous tree, (b) where the large dilatation occurs on the lateral or posterior aspect of the leg, and (c) in that group in which there are recurrent varicosities after operation by a trained surgeon and it is safe to assume that a careful and complete operation of the classic type has been performed See page 589

**Pathology.**—The vein wall becomes dilated The wall loses its elasticity and becomes tortuous The veins may be lifted from their normal position and protrude through the skin

The skin over these veins usually becomes thin, as do the vein walls, and hemorrhage may occur In others, the vein wall thickens due to hypertrophy The veins thus vary from normal to thin, dilated and herniated ones Others have thickened and fibrotic changes following inflammation and intravenous therapy Ulcers are present in 25 per cent

Enlarged veins, when present following a thrombophlebitis, frequently are thicker, appear over a larger surface of the limb, and are followed by ulceration in approximately one year's time The valves will be destroyed in these veins With this valve destruction, the prominent veins are developed with many secondary skin changes Edema is usual, due to ineffective drainage

The pathologic picture present when a small artery connects with the venous system has been described under pathogenesis

**Symptoms.**—The symptoms of varicosities depend upon the site and degree of valve incompetency and vein dilatation, as well as upon the complications In some patients, there are no symptoms, or if present, the symptoms are mild Many of these patients have neglected lesions

The dilatations are most common in the great and small saphenous vein course and along the areas they drain, the medial and posterior side of the leg Above the medial malleolus, especially, occur the secondary skin changes and ulcerations The patient may have dull aching pains in the calves, more evident and intense after prolonged standing This ache is relieved by resting and elevating the legs. When the patient complains of a severe pain and where no phlebitis exists, the likely cause of the pain is not varicosities In more advanced cases, there may be muscle atrophy and weakness.

The legs feel heavy and may swell There is edema around the ankle of the pitting type Nearly all of these patients develop secondary fungus infection With this there is intense itching Many have scratch marks which may be infected secondarily The skin color becomes dusky With ulcerations there is scarring In many the skin is hard tight and shiny particularly over the internal malleolus All degrees of color changes from red to nearly black accompany this process depending upon the amount of pigment (hemosiderin) which has been deposited in the skin

In advanced conditions the pudendal veins may be dilated In the female the varicosities may extend to the vulva and vagina An anastomosis with the pelvic veins here exists Through an anastomosis the veins of the ovaries the uterus, and in the male the scrotum and at times the penis may be involved Other symptoms depend upon the complications that occur

**Treatment.**—Treatment depends upon the condition of the valves the arterial circulation the general condition and age of the patient the presence or absence of an ulcer or other complications the severity of the symptoms and the patient s occupation

Before prescribing the treatment one should determine the status of (1) Arterial circulation (2) Deep vein patency (3) Saphenous-femoral valve competency (4) Incompetent communicating valves (5) The competency of the lesser saphenous system which has valve failures and dilatation in 20 per cent of the patients (6) Other vascular abnormalities

A complete history and physical examination of the patient and the usual laboratory studies help evaluate the best treatment for the individual

1 Arterial Circulation —The condition of the patient s arterial supply is important not because a deficiency contraindicates therapy for the venous problem but because if there is some arterial deficiency the veins must be treated in a manner consistent with the arterial defect Complicated apparatus for testing arterial circulation is not necessary The arterial circulation can be evaluated readily as follows

(*A*) *History* —It is of significance to find out how far the patient can walk and whether when he walks he has symptoms of pain Circulatory pain particularly cramps comes on when a patient is using his limbs thus requiring more blood supply to the muscles than he is able to deliver A pain occurring at night may occur with arterial trouble but a night cramp more often is due to arch or posture defects

A history of susceptibility to infections pressure points or ulcers and infected ingrown toenails which will not heal is important Frostbite diabetes mellitus and undue reaction to thermal changes is significant Color changes in the foot which the patient has noted should be considered

(*B*) *Physical Examination* —The temperature of the foot at various levels is of importance The color of the foot on dependency the rubor which appears when the arterial circulation is failing and the pallor which appears on elevation are significant The presence of pressure points infection ulcerations, absence of hair or trophic changes should be noted

The palpation of the major arteries is of value If the dorsalis pedis and posterior tibial pulses are present there is fair evidence that the circulation is adequate The use of oscillometric readings is of help in the questionable

cases The response of the patient's foot by spasm to exposure to cold or the application of a pressure cuff may be helpful

(C) *Laboratory Methods* —A urinalysis and blood chemistry will rule out the diabetic, the patient with gout, and the nephritic Oscillometric readings, skin temperature readings, and the reaction to antispasmodic drugs will be of value In some, capillary microscopy may be necessary Sympathetic nerve blocks, with observations of the clinical and skin temperature responses, may help to determine the arterial supply The $x$-ray may reveal arteriosclerotic changes not discernible clinically A sedimentation rate and a complete blood count should be performed routinely Arteriography may be required to denote the degree of arterial circulation loss

2 Deep Vein Patency —(A) *History* —The history of a phlebitis or a "milk leg" or frequent attacks of an inflammatory nature will indicate a deep vein closure at a previous time or times The history of postoperative leg trouble (frequent swelling or embolism) should be noted If a patient can walk several blocks with an Ace bandage or elastic stocking without pain or swelling, it is likely that sufficient deep veins are open and functioning for the adequate venous return

(B) *Physical Examination* —(a) The appearance of the leg as to color on elevation and dependency, the measurements of the two legs, the presence or absence of ulceration, and all the other complications of the phlebitic leg may be of help

(b) *"Sentinel Veins"*—Dilatation of three small veins medially over the tibia indicates a deep vein clotting (Pratt Test[19]) See page 626

(c) The appearance of ulcers one year after a phlebitic episode often indicates that the patient had a deep vein thrombosis

(d) *Deep Vein Function Test* [31]—A modification of the old Perthes test is a satisfactory test if there is doubt as to the competence of the deep veins Many such tests have been described The one that we have used for the last fifteen years is described

*Deep Vein Test* —An Ace bandage is applied from the toes to the groin with the foot elevated A tourniquet is placed above the Ace bandage, sufficiently tight to close off the superficial venous supply The patient walks for ten minutes

Careful circumferential measurements of the legs and feet before and after the walking should be recorded The bandage and tourniquet temporarily have closed off the superficial venous return If the deep venous circulation is inadequate there will be pain and swelling

The measurement of the legs may be made by water displacement The leg is placed in a colonic pail partly filled with water, and the level of the water is measured After the patient has walked with the Ace bandage and the tourniquet in place, this is repeated The difference in water level measures the increase or decrease in leg size

(C) *Venography* —With experience, venographic tracings can be interpreted to denote accurately deep vein patency

(a) Radiopaque solution (30 per cent Urokon) is injected into a vein over the dorsum of the foot usually near the great toe A tourniquet around the ankle prevents it from ascending the superficial veins The deep veins may be delineated well in this way Incompetent perforating veins with

valve failure will be demonstrated by a leak from the deep vein into the superficial system above the tourniquet

(b) With the patient standing the common femoral vein may be injected If the dye runs distally into either the superficial femoral the saphenous or the profunda vein such veins have incompetent valves Secondary leaks can also be determined in this manner This method may be modified by injecting the dye directly in the femoral vein on the operating table and then lowering the foot of the table while the head of the table is elevated The x ray technic includes exposure at 1/10 of a second with between 55 and 74 kilovolts individualized for the patient s size The cooperation of the surgeon and the roentgenologist as to timing is most important. Experience is required in the x ray interpretation With such experience however this method of study best determines the patency and adequacy of the deep veins and their valves

(D) *Test for Deep Vein Function at Operation Time* —It has been found that if the superficial vein is incompetent and there is an adequate profunda occlusion of the superficial vein results in this vein shrinking and closing off If the profunda is inadequate however and the superficial femoral vein is necessary for venous return its occlusion causes it to dilate distally At the operation time therefore temporary occlusion of the superficial femoral vein will demonstrate these facts If its occlusion causes the vein to become smaller it may be divided with impunity If such occlusion results in its dilatation below the occlusion point it should not be ligated

3 Saphenous-Femoral Valve Competency —This is a modification of the Brodie-Trendelenburg test The limb is elevated and a tourniquet is then applied The patient stands up and the tourniquet is released Normally the circulation should fill the veins *from below up* in thirty seconds If the circulation fills rapidly *from above down* it is evident that there is a leak from the femoral circulation into the saphenous vein through the saphenous-femoral valve Such a leak is present in 85 per cent of our patients who have varicose veins. This can be seen grossly and the experienced observer can demonstrate it by holding the blood column back with the fingers The venogram also demonstrates incompetency of this valve

4 Incompetent Communicating Branch Valve Test While an adequate operation for varicose veins requires a thorough resection of the saphenous vein and its branches in the groin such an operation alone is not sufficient The leaks from the femoral vein through the communicating branches to the saphenous system at other points also must be resected The determination of such points and their marking prior to operation is of fundamental importance Many tests have been devised for the determination of such defects These defective valves should be marked in the operating room just before the operative procedure We use an indelible pencil for this purpose Other marking substances have been devised such as the dyes etc [16] Because of the difficulty in interpreting other tests our Clinic has used the following test for over seventeen years

*Pratt Test* [17]—The limb is elevated emptied of blood and Ace bandages are applied from the toes up to the groin A tourniquet is then applied

above the bandage The tourniquet temporarily acts as a high saphenous ligation, shutting off the incompetent femoral-saphenous leakage The patient then stands up and the Ace bandages are unwound from above down, leaving the tourniquet in place If a blood vessel pool is seen at one point and can be pressed back with one finger, but recurs like a hernia when the finger is released, it indicates that at that point there is a leak from the femoral vein to the saphenous system Such a blood vessel is

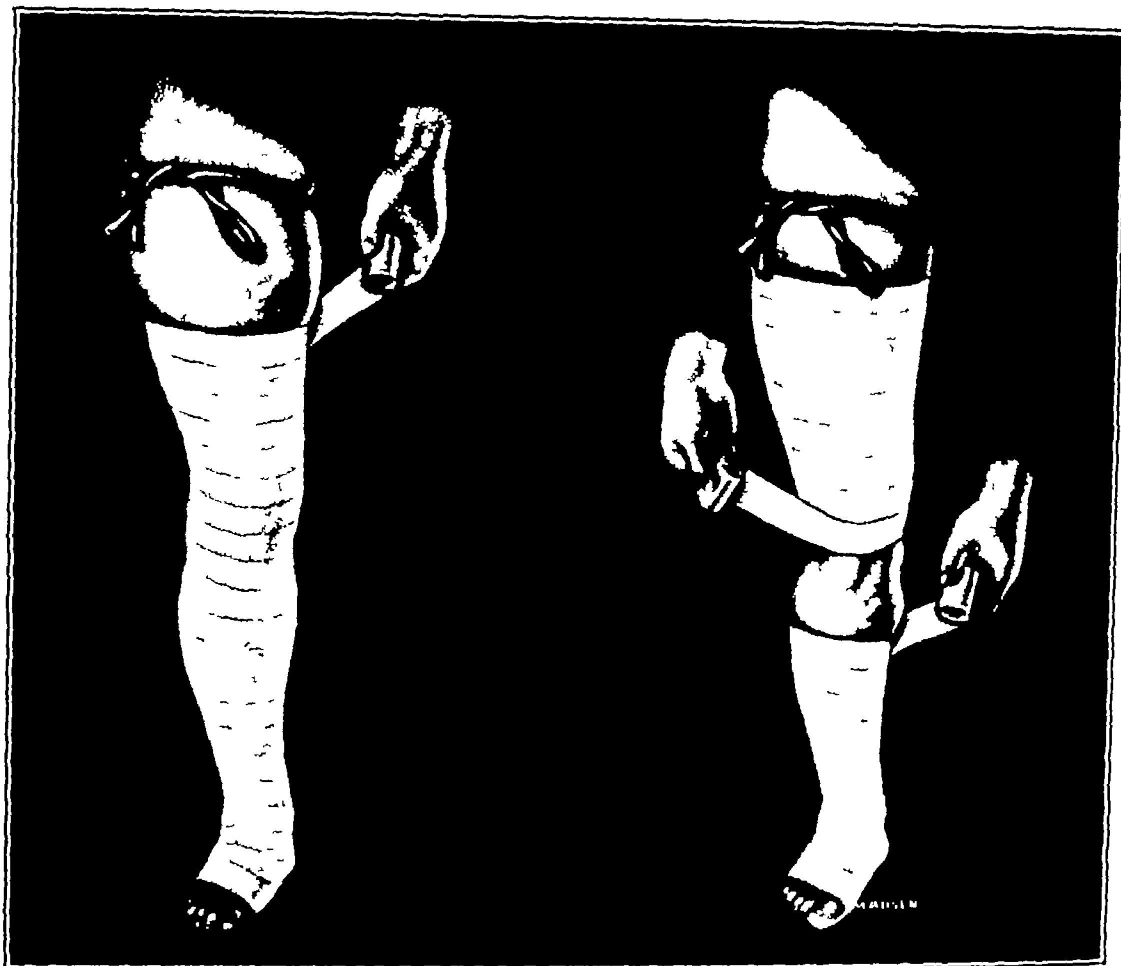


FIG 172 —Pratt Test for incompetent communicating points In this test the veins of the leg are emptied by elevation Ace type bandages are applied from the toes to the upper thigh and a tourniquet is applied The patient stands and the Ace bandage is then unwound A collection of veins at any point shows the incompetent point A second Ace bandage may be started and wrapped from above down so that only a small area of the leg is free of bandage at any time (*Pratt, courtesy of J A M A*)

unable to fill from below because of the pressure of the Ace bandage, and the tourniquet prevents a leak from the femoral vein above.

As the Ace bandage is unrolled, another is applied from the top down, so that at no time is there more than a hand's breadth of leg area exposed Each incompetent point can be checked and marked in this way This is done just prior to operation and each incompetent point must then be resected With experience, the second Ace bandage is not required One can feel the hole in the fascia through which the incompetent perforator

communicates We have had such good results with this test that we have discontinued using the Perthes test the percussion test of McCallig and Heyerdale[12] the comparative tourniquet test of Ochsner and Mahorner[17] and others The benefits of these tests have been described by their originators and their value is not decried We have been unable to interpret the results of such tests as accurately as the one described above

5 AUTHOR'S TEST FOR INCOMPETENT VALVES BY MANUAL METHOD —In experienced hands the blowout points can be identified by the hands only See Fig 174 A and B This method has been used by the author for eighteen years

6 VENOGRAPHY —As described above venography by the technic outlined may show the sites of incompetent communicating valves. This is needed rarely in simple varicose veins

7 OTHER TESTS FOR VASCULAR ABNORMALITIES —In some patients oxygen saturation tests are necessary to determine the presence of arteriovenous fistulas Skin temperature readings may also indicate an area of arteriovenous fistula Careful auscultation for aneurysm should always be made Roentgen ray examination of the limb sometimes is necessary to rule out calcification Large venous collections and phleboliths can be identified

*Venography for Incompetent Perforating and Superficial Veins* —Venography has been used for many years With a better standardized technic, its value has been augmented One can determine the sites of valve incompetency in the superficial and communicating branch systems The adequacy of the deep vein and its valves also is demonstrable by this method In the questionable case one can avoid the disasters which follow the resection of a spahenous system in the presence of a poorly functioning femoral vein return by this method In other patients the superficial femoral vein can be injected with a dye on the operating table, the foot of the table depressed and the function of the femoral vein valves visualized

**General Rules for Therapy** —While no specific rules for the treatment of all cases of varicose veins can be drawn up certain general rules apply

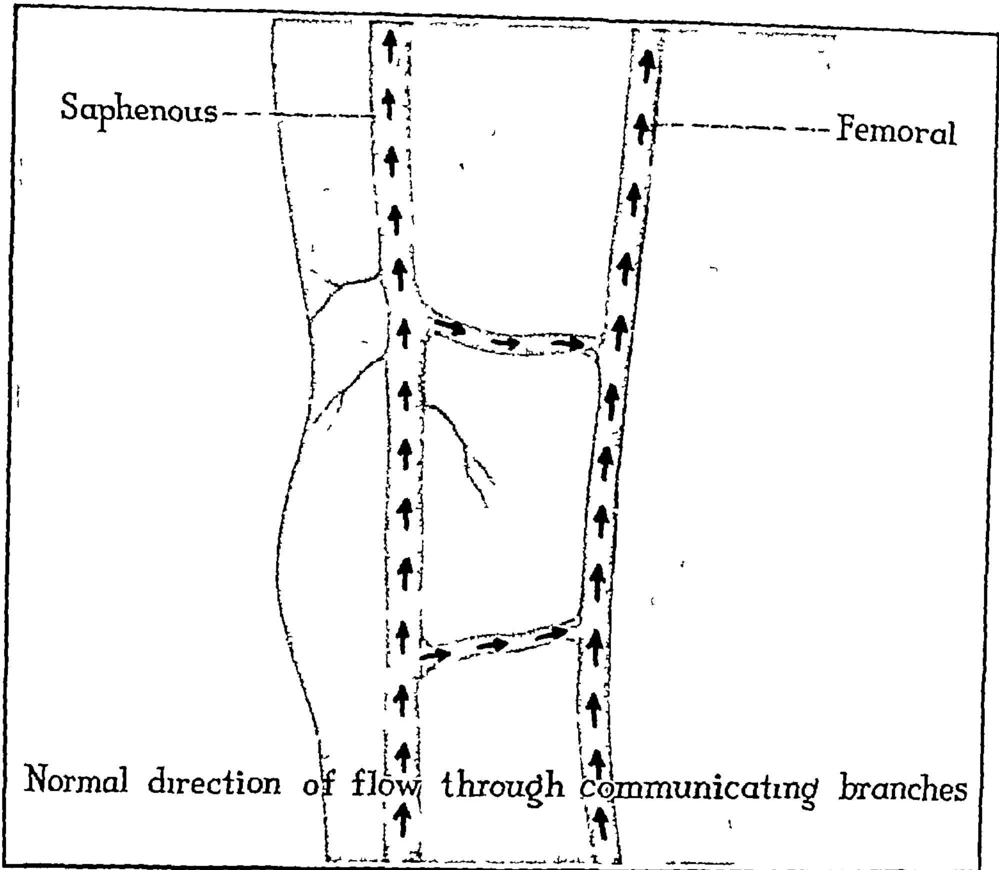
(1) Competent saphenous-femoral valve and communicating branch valves but dilated veins *Treatment* Local injections This applies only to 8 to 15 per cent of our patients

(2) Incompetent saphenous-femoral valve with competent communicating branches *Treatment* Resect the saphenous vein at the femoral junction with all of its branches and strip the saphenous vein between the incompetent points The lesser saphenous vein also should be resected

(3) Incompetent saphenous femoral valve and incompetent communicating branch valves. *Treatment* Resect the saphenous vein high with all its branches resect widely each incompetent point and strip between the points The lesser saphenous vein also should be resected

(4) Incompetent saphenous-femoral valve and incompetent communicating branch valves plus ulcer *Treatment* When ulcer is clean operate as in number 3

(5) Incompetent saphenous-femoral valve and incompetent communicating branch valves after phlebitis *Treatment* If phlebitis is not active resect as in number 3 Such an operation is best delayed for a minimum time of six months after phlebitic activity



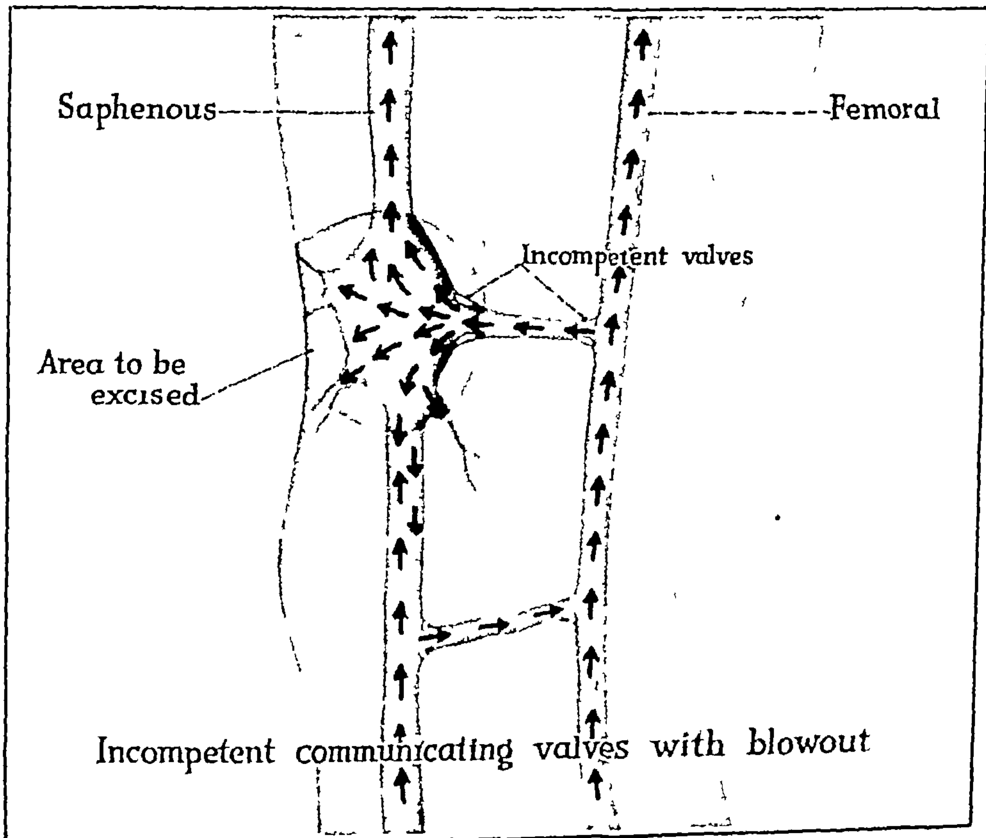




FIG. 173 —Diagrammatic drawing of the way an incompetent valve causes a "blow-out." Arrows show the normal direction of flow from the saphenous to the femoral system. Lower picture shows the reversal of flow when the valve fails. (Pratt, courtesy of J. A. M. A.)

(6) Incompetent saphenous femoral valve and incompetent communicating branch valves after femoral phlebitis with ulcer plus competent femoral profunda *Treatment* If superficial femoral vein and its valves are incompetent resect superficial femoral vein and resect saphenous system as described in number 3 In all cases, the adequacy of the deep vein should be determined by venography and clinically and confirmed at the operating table




FIG 174.—Pratt Manual Test for incompetent points Shows author's test for incompetent points performed manually In this instance the large veins are reduced as one does a hernia and held with the two fingers and a second incompetent point can then be demonstrated and marked.

(7) Inflamed vein with or without incompetent valves. *Treatment* Conservative management. No operation is advisable unless there is uncontrolled extension or embolism

**Contraindications to Varicose Vein Operation.**—In our experience any active infection or inflammation contraindicates operation on a vein This includes general or local phlebitis a cellulitis or a suppuration in an ulcer Some clinics operate on patients in an inflamed state and report good results Our experiences have been exactly the opposite and we try to have our

patients in a non-active state before prescribing operation The only exception to this rule is where there is progression of the process or embolism despite conservative management In addition, no operation would be performed on a superficial vein if the deep system was inadequate for venous return See tests for deep vein patency, page 564

**Local Injection Treatment of Varicose Veins.**—Chemical sclerosis treatment is satisfactory in cases of simple vein dilatations without major valve damage

In order successfully to obliterate varicose veins chemically, it is necessary to irritate the vein, produce a clot, and maintain this clot's position until fibrosis has occurred[4,5,25] This is done by using some solution which is destructive to the intima The solutions generally used for this purpose are (1) A hypertonic solution of concentrated glucose or salt, which causes dehydration by osmosis (2) Strong alkaline solution, such



Fig 175 —Reaction vessel wall to a sclerosing solution at the site of injection two weeks after the injection (Pratt, courtesy of Am J Surg )

as one of the soap solutions, sodium morrhuate, sodium ricinoleate, or sodium licinoleate, which destroys the intima wall (3) Substances such as quinine hydrochloride or bichloride of mercury, which act by poisoning cell protoplasm

The rate of action of these substances varies Bichloride of mercury causes clots in three days, whereas strong salt solution is effective in one day The concentration of the solution controls the effect

The sclerosing effect depends upon the following five factors (1) The amount and strength and type of solution (2) The condition and size of the vein injected (3) The dilution of the irritant by blood in the vein or its collateral circulation (4) Stasis or activity after the injection (5) The reaction of the patient's tissues to the injected solution

No tourniquet should be used during the injection treatment A tourniquet temporarily may retain a clot in the vein, and when the tourniquet is released, the clot then may move The injections are begun in the highest visible vein in the thigh, and then repeated from above down Small

amounts of sclerosing solution at frequent introduction points give a more satisfactory fibrotic effect than a large amount at one place See Figure 177 A pressure bandage is applied after the injections and maintained for twenty-four hours.

TABLE 42 —TYPES OF SCLEROSING SOLUTIONS

*Group I* —Hypertonic solutions Effect dehydration by osmosis irritation of vein intima Sugars and Salt

| *Chemical* | *Active principle* | *Amount* |
|---|---|---|
| Glucose 50% 30% | Dextrose | 5 10 cc |
| Sodium Chloride 30% 20% | Salt | 5 cc |
| Glucose 30% plus Sodium Chloride 20% | Sugar and Salt | 5 cc |
| Sodium Salicylate 20% | Salt | 5 cc |
| Levodex | Invert sugar | 5–10 cc |
| Varisol | Invert sugar—3 grams<br>Salt—1 gram<br>Benzyl carbinol—0 1 gram | 2–4 cc. |

*Group II* —Soaps or soap-like substances

| *Chemical* | *Active principle* | *Amount* |
|---|---|---|
| Sodium Morrhuate 5% | Sodium Morrhuate 5%<br>Benzyl Alcohol .2% | ½–2 cc |
| Sodium Ricinoleate 5% | | 2 cc |
| Sodium Licinoleate 5% | | 2 cc |
| Monolate 5% | Monoethanolamine oleate<br>{Monoethandamine oleate 5%<br>Benzyl Alcohol 2%} | ½–2 cc<br>Total 2 cc |
| Sylnasol | 5% Solution of the Sodium Salts of fatty acids of Psyllium oil and Benzyl Alcohol 2% | ½–2 cc |
| Sotradecol 1%<br>3%<br>5% (rare) | Sodium tetradecyl sulfate (2-methyl-7 ellylundecyl-4 sulfate)<br>2% benzyl alcohol | 1% for Spider type varix. .2–2 cc.<br>3% ½–2 cc. at multiple sites. No more than 2 cc total.<br>5% single dose ½–2 cc Total no more than 2 cc |

*Group III* —Tissue cell cytoplasm poisons

| *Chemical* | *Active principle* | *Amount* |
|---|---|---|
| Quinine Hydrochloride 5% | Quinine Hydrochloride 5% | ½–2 cc |
| Bichloride Mercury 1 1000 | 1 1000 solution Bichloride Mercury | ½–2 cc |
| Quinine Hydrochloride & Urethrane | Quinine Hydrochloride 0.266 gm (4 gr)<br>Urethrane 0 133 gm (2 gr) | 2 cc |
| Quinine & Urea Hydrochloride 5% | Quinine & Urea Hydrochloride 5% 0 1 gm (1½ gr) | ½–2 cc. |

If injection sclerosis is to be used, the solution should be injected uniformly throughout the vein rather than by a needle at a single point The necessity of introducing the solution uniformly and at many points of maximum intensity is illustrated by the photomicrographs shown in Figures 175 and 176

Figure 175 shows the sclerosis in a vein of a rabbit's ear at the point of introduction of injection two weeks after the injection

Figure 176 shows a section of the same vein 2 inches from the point of injection and indicates that the wall has not been affected

The degree of sclerosis of the veins thus is inversely proportional to the distance from the point of injection

These sclerosing solutions are injected best with the patient standing Only 1 cc of solution is used the first time to note the patient's reaction to the drug



Fig 176 —Shows same vein as Fig 175, two inches from injection point, with very little reaction to the solution (Pratt, courtesy of Am J Surg )

Patients with incompetent saphenous femoral valves, whether they have incompetent branch valves or not, require surgical treatment if the desired effect is to be obtained Sclerosing injections in such patients are not only ineffectual but compound the picture by causing the formation of other collateral veins

**Equipment for Local Injections.**—The standard vein injection equipment consists of 25-, 26-, or 27-gauge short beveled needles, 1½ inches long, and a well-fitted 2 cc syringe.

**Technic.**—The patient should stand on an elevation or table with a hand support in a good light The operator should be seated, preferably at the level of the patient's knee It is well to have a support for the operator's elbow so that the needle may be held steadily See Fig 177

**Response.**—The response to local injection varies with the dilution of the solution after it is injected When the solution is injected into a large vein it may be diluted as much as 100 times, while in a small vein the dilution may be only a few times This accounts for the severe sclerosis in

the small veins and at times, sloughs. With experience, one can estimate readily the correct quantity of solution for each vein both by the vein's size and the distention which occurs during the injection. This dilution factor may be utilized to produce greater sclerotic effect by milking out the blood in a large vein prior to the injection. The sclerosis is local and the greatest effect is at the point of injection.

The patient should be ambulatory after the injection. The incidence of pulmonary embolism after these injections is approximately 1 in 15,000



FIG. 177 —Local injection technic. Patient is standing with a support for hands and in a good light. The doctor is seated before the patient and at the right level. A short beveled 25- 26- or 27-gauge needle is used.

but this is multiplied 6 times if the leg is put at rest after the injection due to the excessive clotting which may occur with stasis.

**Contraindications to Injection Therapy.**—The following conditions are contraindications to injection therapy (1) deep vein incompetence (2) acute thrombosis or thrombophlebitis (3) cellulitis or local infection (4) uncontrolled diabetes mellitus (5) incompetence of the spahenous femoral valve and communicating branch valves (6) ulcerations which are infected or inflamed.

**Varicose Veins and Arterial Deficiency.**—Heart disease and advanced arterial deficiency are no longer considered contraindications to a vein

elimination, although greater care must be taken in the injection of such patients In the presence of an obliterative arterial disease, reflex arterial spasm may occur after any vein trauma, including a vein injection The quantity of solution used in these individuals should be small Preparations must be made to counteract any general reaction or arterial spasm by the

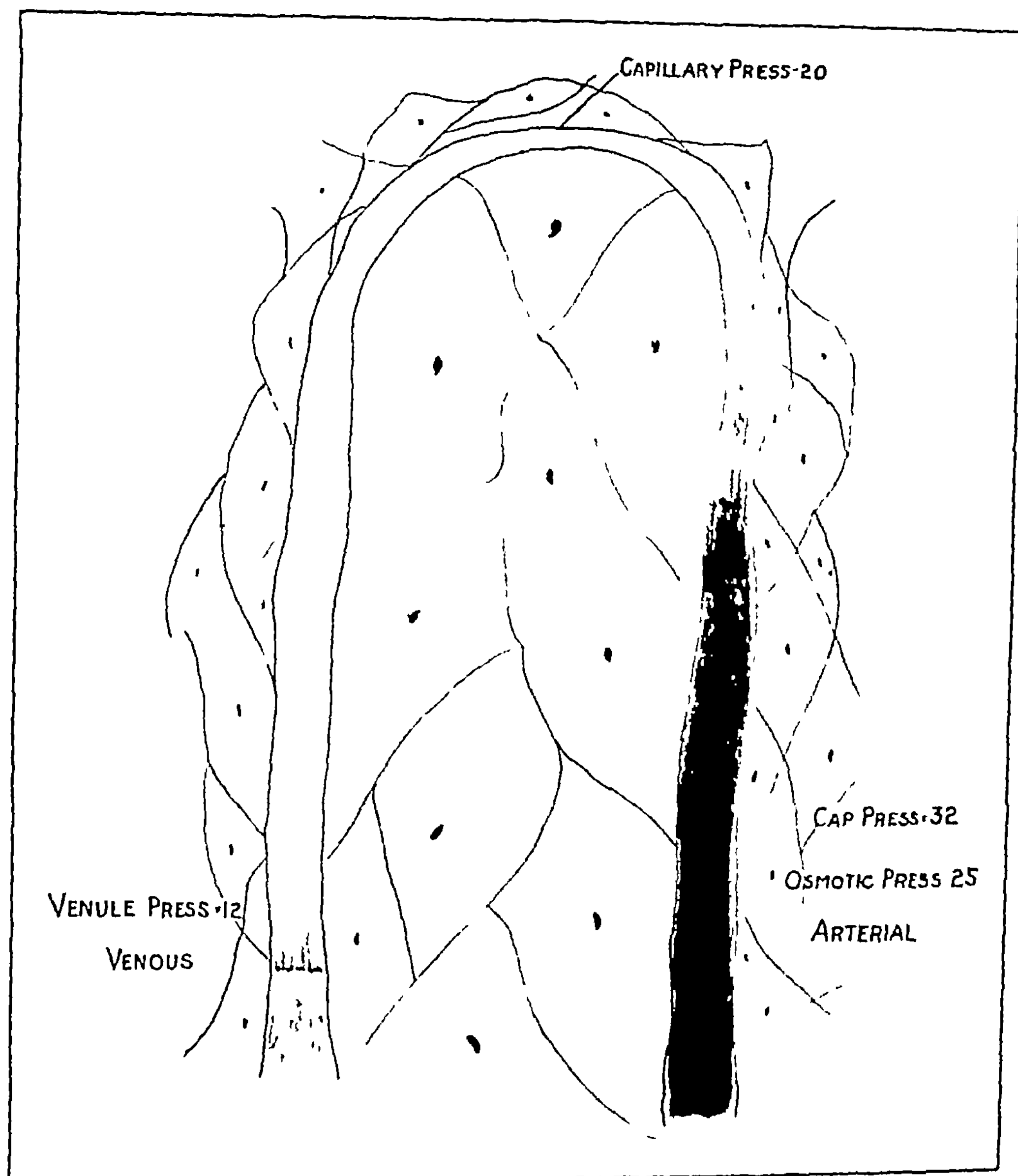



*Fig* 178 —Normal capillary loop (revised from Landis) The positive pressure within the loop decreases as one approaches the venous end while the osmotic pressure increases, thus aiding in the exchange of fluid between the loop and the tissues If the venous pressure is increased as when there are varicose veins, the pressure against which the arterial blood must flow is greater, and vice versa

methods outlined under the conservative treatment of Occlusive Arterial Diseases on pages 162 to 176

For many years arterial occlusion in a limb was considered a contraindication to the treatment of pathologic veins It was the belief that nothing should be done to any part of the blood supply in such a limb on

the theory that excessive venous blood was better than no blood at all This concept is no longer valid

In the capillary bed, arterial and venous blood mingle There is arterial blood at one end and venous blood at the other end of each capillary (See Fig 178) Dilated veins with incompetent valves greatly increase the venous back pressure This increased venous pressure is transmitted back on the venous end even as far as the capillary loop This increases the capillary bed pressure against which the blood in the already depleted arterial supply must work

According to Landis the capillary pressure at the arterial end of the capillary loop is plus 32 with an osmotic pressure of minus 25 There is therefore a positive outward pressure At the venous end of the loop the positive pressure is plus 12 The relatively high osmotic pressure therefore draws fluid into the loop and physically promotes the intracellular capillary fluid exchange If the venous pressure is higher due to valve incompetency above the venule level the exchange is hampered and greater strain is placed upon the already depleted arterial supply Removal of the deficiency at the venous end will help the general circulation

The judicious removal of the pathologic veins in the presence of an impaired arterial circulation will aid the arterial circulation by reducing the peripheral resistance We have performed operations many times specifically to aid this arterial impairment in patients with pathologic veins and have seen improvement in the arterial symptoms Similar results have been achieved in the cardiac patient where the cardiac status is improved by the removal of these stagnant venous pools the effect being comparable to the removal of a pleural effusion

## SURGICAL TREATMENT OF VARICOSE VEINS

Where the saphenous femoral valve and/or communicating branch valves are incompetent operative therapy is necessary Surgical treatment can be performed whether there are open ulcers present or not but the ulcer should not be infected

*Contraindications* to surgical treatment of varicose veins are (1) active phlebitis (2) local infections (3) an inadequate deep femoral vein system and (4) uncontrolled systemic diseases such as cardiac decompensation and diabetes mellitus These conditions should be corrected before operation is contemplated

The operation should be performed only in a hospital because this procedure may become complicated Excellent assistance is required Extra care is necessary in the cleansing of the primary operative site because this area near the vulva and scrotum is prone to infection The entire leg groin vulval or scrotal area should be shaved and washed We have discarded all other preparations except sulfonated detergents (pHisoHex) and sterile drapes The wound complications since that time are rare

**Ligation and Retrograde Sclerosis of the Saphenous Vein.**—This operation which was the outgrowth of the work of many surgeons was the operation of choice for the period from 1935 to 1945[3,7,11,12,25] It has now been replaced in nearly all clinics by ligation, resection of incompetent

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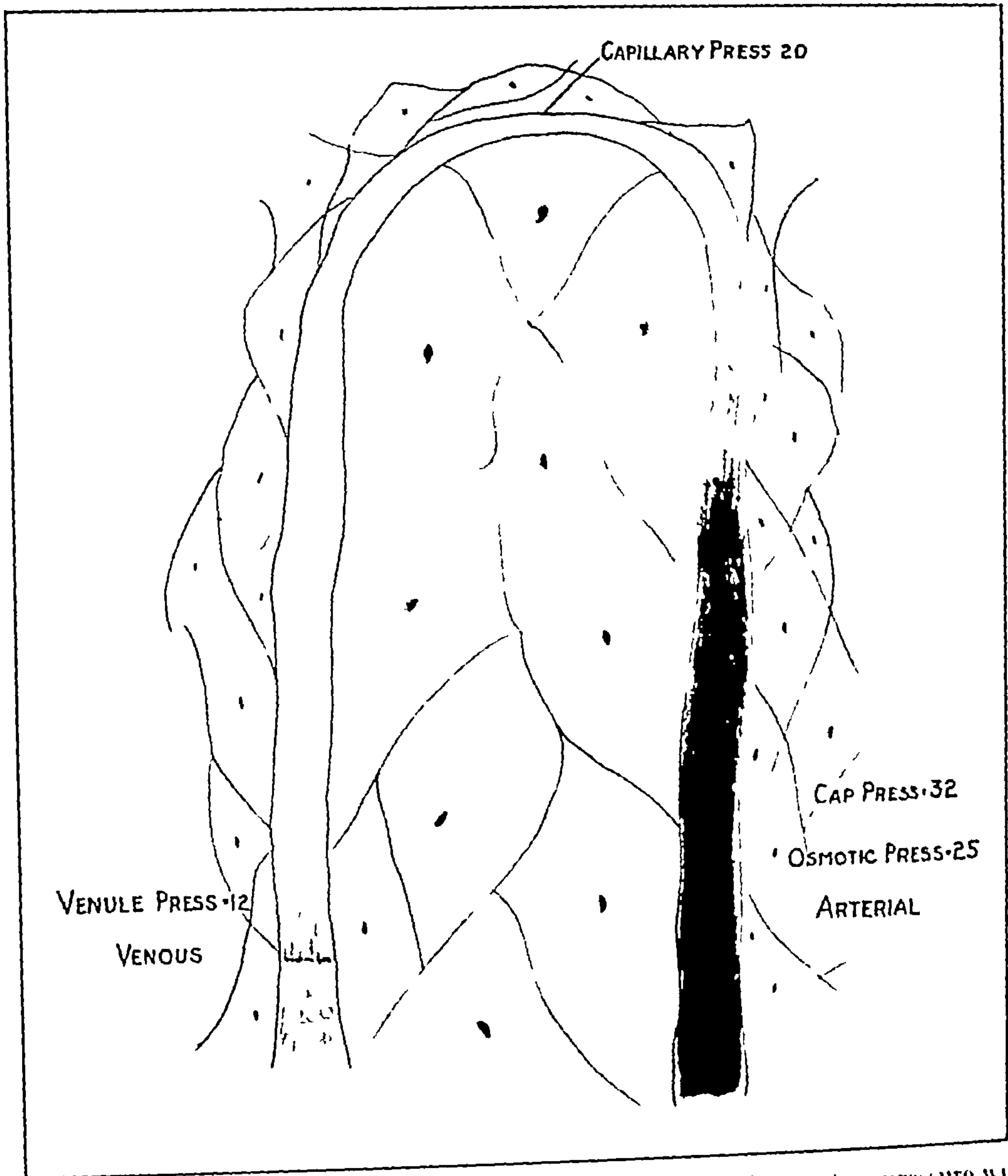



FIG 178 —Normal capillary loop (revised from Landis) The positive pressure within the loop decreases as one approaches the venous end while the osmotic pressure increases, thus aiding in the exchange of fluid between the loop and the tissues If the venous pressure is increased as when there are varicose veins, the pressure against which the arterial blood must flow is greater, and vice versa

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**Ligation and Retrograde Sclerosis of the Saphenous Vein.**—This operation which was the outgrowth of the work of many surgeons was the operation of choice for the period from 1935 to 1945 [3,7,11,14,23] It has now been replaced in nearly all clinics by ligation resection of incompetent

points and stripping. When used, the procedure consisted of a resection of each branch of the saphenous vein in the groin, a resection of each incompetent point and a retrograde sclerosis with a sclerosing solution in the remaining superficial vein. In the technic we followed, this solution was injected through a sterile ureteral-type catheter as the catheter was withdrawn from the vein.[24,25] Complications to this procedure included a chemical reaction, an occasional slough, pain and greater recurrences than



Fig. 179.—Extensive varicose veins with secondary skin changes. Veins of this size often have arterial connections. Patient's syncope caused by blood loss to his legs when he stood up was cured by resection of vein pools.

followed the newer technics. This operation is advocated today only for those patients in whom for some reason the more modern procedure cannot be performed.

*Dangers of Ligation and Retrograde Sclerosis.*—In reviewing the results of these patients over a period of ten years, in 1946 it was found that about 60 to 70 per cent had a satisfactory result.[23] Twenty per cent had incompetencies of the lesser saphenous vein. Approximately 15 to 20 per

cent had new incompetent points The patients who had arterial connections or undiagnosed phlebitic lesions were considered errors in diagnosis This procedure became complicated at times even in the hands of well-trained surgeons It is apparent that if an operation of this type is left to hospital interns and inexperienced surgeons there will be accidents and in a certain number of cases serious ones

A summary of some of the disasters following the operation of ligation and retrograde sclerosis of varicose veins was compiled by Luke and Miller[15] These investigators listed 20 disasters and the reasons for their occurrence These included three deaths three amputations in others severe pulmonary emboli and massive edema Analyzing these reports, it appears that many of the accidents were anatomical errors Most of the reported incidences of gangrene could have resulted from the ligation or sclerosis of the artery rather than the vein The artery will not pulsate when it is in spasm and its superficial nature may make it appear to be a vein The importance of anatomical study before any surgical procedure by all surgeons no matter how experienced is again emphasized The following operations have replaced this procedure

## RESECTION AND STRIPPING OPERATION FOR VARICOSE VEINS

*Anesthesia* —Spinal anesthesia is the anesthesia of choice for this operation Resection can be done under local anesthesia but inasmuch as many of the veins are attached directly to the skin there will be some pain in the stripping if only local anesthesia is used The anesthesia should be limited to the legs

*Operative Technic* —The patient is examined just before the operation and the incompetent points are marked off See pages 565 567 Through a transverse incision centering over the fossa ovale and located 1 inch below and medial to the spine of the pubis the saphenous vein and all of its branches are dissected free and widely resected The branches are resected for a distance of one inch from the saphenous vein itself at the femoral junction The saphenous vein is ligated with a transfixion suture of 00 plain catgut at the femoral junction Separate incisions are made in the line of the vein at the marked areas and each incompetent communication branch point then is widely resected

A modified Babcock type (intraluminal) stripper is introduced into the distal divided saphenous vein retrogradely or occasionally from below up At this stage the stripper becomes a diagnostic instrument. Sometimes it will be found that the stripper passes the site of the secondary incisions. This indicates that the veins dissected by the assistant are only branches of the saphenous and not the main vein at all The stripper thus is used to remove the missed pathology It is left in place until the veins are dissected down to the vein the stripper has entered At other times the stripper may demonstrate another equally pathologic branch which requires resection. When introduced from below up the stripper may aid in the groin dissection where identification of the veins is difficult due to adenitis scarring multiple veins or previous operations In the secondary operation this measure has great value For stripping the vein is tied in two areas

$\frac{1}{8}$ of an inch apart around the acorn enlargement with a #1 catgut suture and the stripper is then pulled through A steady continuous downward pull works best The vein usually piles up on the stripper in an accordion-like fashion and comes out in one section At times, the vein may turn inside out and come out inverted The pull on the intraluminal stripper should be a slow, steady continuous one The pull should be just sufficient to move the stripper without pulling it loose from the vein It can be likened to the pull required to land a fish A jerk or fast tug will break the vein off just as it will lose the fish The right degree of tension delivers the vein like it lands the fish Stripping is accomplished after each communicating branch point has been isolated It is necessary to perform the same procedure on the lesser saphenous vein

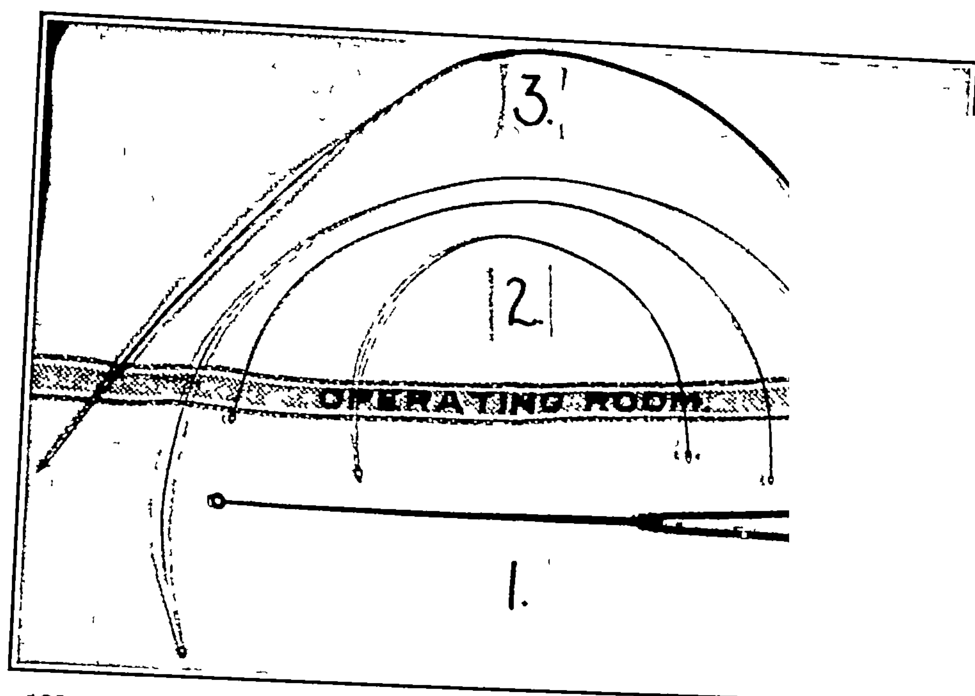



FIG 180 —Various types of vein strippers 1 Extravenous strip
2 , 3 Intraluminal-type stripper with various size acorns on the end o
cock type )

If there is difficulty in introducing the stripper, it is an r
often that there is a communicating branch point which ha
At such times, a second incision at the point of difficulty sh
and this area resected The stripper will aid in the location
these secondary incision points On many occasions it has be
the stripper passes by the site at which the second team n
incompetency below the knee It is apparent, therefore, th
superficial varices above the blowout point were resected ra
actual site of saphenous incompetence The stripper has a
function, therefore, to demonstrate such technical errors I
a supplementary or secondary vein in addition to the obviously
vein A malleable guide has been used for this purpose T
technic is not a return to the older methods of treating varic
merely stripping In the older procedure, a simple incision and

was done usually at Trendelenburg's point (junction middle and upper third of the thigh) without resection of the vein and its branches at the junction with the femoral vein Incompetent communicating branches were not resected These communicating branches tore off in the stripping and then recurred by a wide anastomosis in a short time

When the operation has been done adequately and recurrences are prominent one must suspect and look for small arterial connections to the veins This is more common than has been suspected In those with veins which do not completely empty on elevation or where there is rapid and enormous dilations of the veins or where they recur after adequate operation one always should suspect arterial connections See Arterial Varices pages 589 to 595

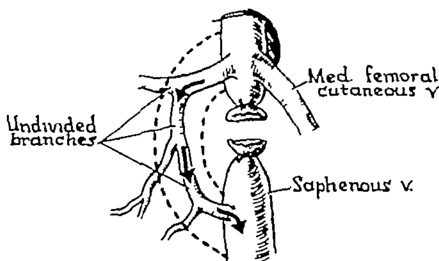



FIG 181 —Drawing illustrating method of recurrence of varicose veins when a branch in the groin is undivided. Dotted lines show dilatation which will occur

*Treatment of the Incompetent Points by Resection* [24]—At each incompetent point as marked before operation an incision is made These incisions are best made vertically in the line of the vein's course as a transverse incision may miss the feeding or perforator vein Where the incompetency occurs at a bifurcation of a vein the incision should be between the two branches so both can be resected It is best to widely expose and resect these points as there may be more than one perforator vein Each incompetent perforator must be widely resected with the "blowout." It is most important to widely resect all branches of the saphenous vein at the internal malleolus

*Author's Suspension of the Distal End of the Vein after Blowout Resection* —Where the veins are tortuous and widely dilated at the ankle they may be suspended after the resection The "blowout" and all branches are resected as before and the perforator divided and ligated The distal end of the incompetent area may be transfixed and ligated and then sutured to the deep fascia on tension This straightens out the tortuous vein and provides a suspensory ligament for the vein and the surrounding tissue like in a varicocele resection and suspension operation Where this

Fig 182 —Babcock type vein stripper with vein Intraluminal stripper

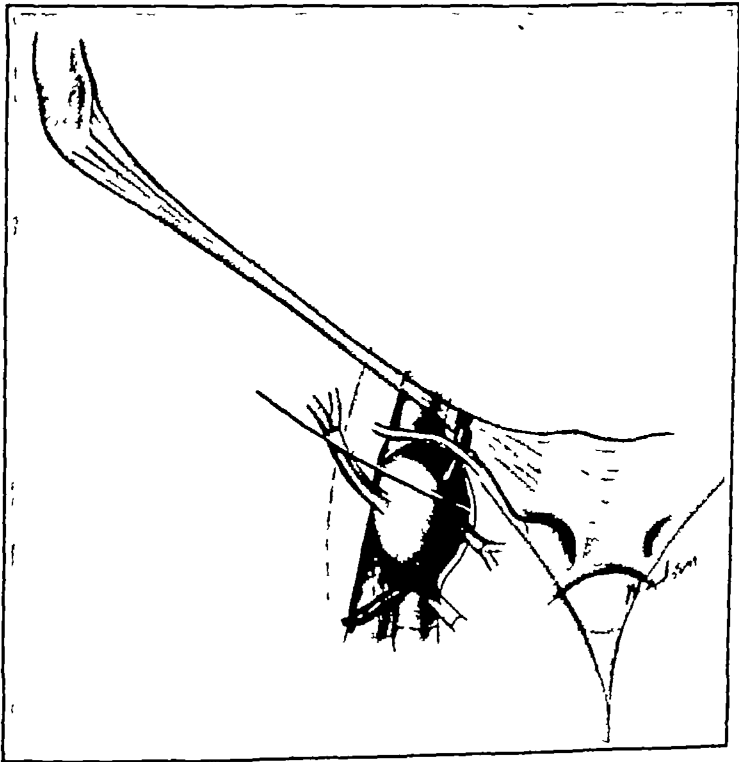


Fig 183 —Anatomy of saphenous femoral junction The superficial pudendal artery marks the anatomical division of the saphenous and femoral veins (Pratt courtesy of J, A, M, A )

is just above an ulcer or above diseased skin, this procedure relieves edema and aids in healing

**Other Technics** —*Extra Venous Strippers* —The loop stripper which fits outside the vein was invented by Mayo[14] There are places in which it can be used but these instances are rare This stripper is traumatic often

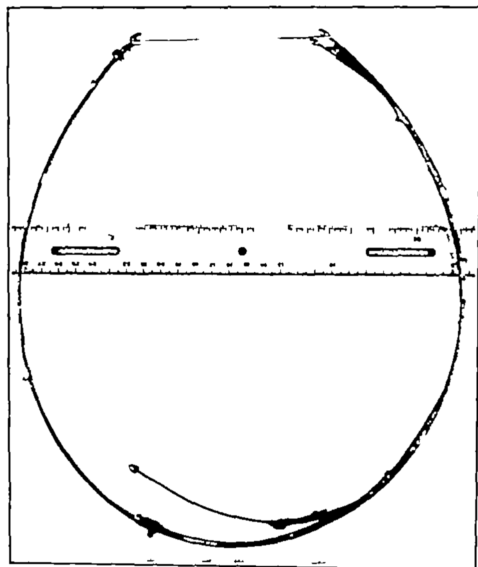


Fig 184 —Author's multiple stripping technic. Several strippers may be used to demonstrate accessory incompetent veins This technic eliminates the large accompanying veins which may permit immediate recurrence

tears the main vein or its branches and its prime use is to remove segments of vein which have been diseased and have no lumen

*Other Strippers* —Malleable strippers have been used These are made of soft metal, chains or wire. We prefer a firm wire stripper as it helps find other branches better

*Retrograde Stripping* —Some surgeons use probes passing these instruments down to the first branch. This is but a variation of the routine stripping[37]

*Multiple Stripping Technic*.—Both in the groin and at incompetent points, more than one vein of equal size may be encountered. All of these veins can have strippers introduced at the same time. This identifies other branches which would be missed. At times, 4 to 5 strippers are placed in veins at the same time. These may be pulled out simultaneously or at different times dependent upon anatomical positions. Often it is necessary to make a secondary incision at the site where one stripper joins another. Whether to strip up or down again depends upon local anatomical conditions. In general, we strip down against the valves. If this is impossible technically, or if the vein breaks, the process is reversed.

**Postoperative Care.**—The opening in the groin wound left by the stripped saphenous vein should be closed by a figure of eight fine catgut suture. The bandaging, otherwise, will force blood along the stripped vein's tract and cause a hematoma to develop in the groin wound.

The wounds are closed with interrupted #35 or #36 steel wire sutures. These sutures have little or no tissue reaction if they are not tied tightly. They may be left in place as long as two weeks and are particularly valuable if there is some drainage. They do not have to be removed in such an event as silk sutures would require, and the separation of the wound edges thus is prevented. An adequate padding and bandaging are required to prevent a hematoma. Postoperative care following the stripping operation should include passive motion of the limbs every fifteen minutes until the spinal anesthesia has worn off (six hours), at which time active ambulation is begun. The patient walks fifteen minutes out of every two hours. There has been no greater incidence of spinal headaches from this early rising. We believe that spinal headaches do not result from the anesthesia or walking but follow the excessive loss or removal of spinal fluid. In the modern technics, with #26 gauge needles where not more than 1 or 2 drops of spinal fluid are lost, headaches can be discounted.

**Complications of Varicose Vein Operations.**—(1) DUE TO INADEQUATE SUPERVISION.—From the frequency with which complications occur following operations for varicose veins, it is apparent that in many hospitals, the treatment of varicose veins is considered secondary in importance and is frequently relegated to the hands of new and inexperienced house surgeons. In certain institutions the procedure has been made an outpatient procedure.

This is not safe. This vein operation, which usually is simple, may become extremely complicated at any moment. Accidents have resulted in a fatality or the loss of a limb.

The work should be under the direction of surgeons trained in vascular work who are interested in it and will give to it the time required to obtain a good result. This is important, not only for the safety and success of the procedure but because if the operation is poorly or inefficiently done the first time, so much pathology will be developed from the operation itself that correction thereafter will be extremely difficult or impossible.

(2) LIGATION OR INJURED ARTERY.—The femoral artery has been ligated, injured and even sclerosed many times. This error has been made due to mistaking it for the saphenous or femoral vein. The artery in the groin is superficial and at times may not pulsate vigorously due to spasm. It is

in intimate association with the vein at the saphenous-femoral junction The accident occurs usually when there is some hemorrhage which conceals the anatomical vascular arrangement The bleeding from a torn saphenous vein can be profuse and the inexperienced surgeon may place his clamp too deep In addition the artery has been considered to be the vein too often and with too serious consequences to be taken lightly Chiefs of Service have not been exempt from this error We know of 18 instances in which the femoral artery, instead of the femoral vein, has been divided

(3) HEMORRHAGE.—The varicose vein is notably friable extremely thin-walled tortuous and may be torn readily This is particularly true as one approaches the femoral bulb It is also true of the varices or small branches which must be resected near the bulb Adequate exposure and careful retraction will prevent these tears

Since these branches are particularly thin they should be dissected free with a hemostat and the proximal end ligated prior to dividing it

This eliminates the danger of the clamp slipping or the suture breaking with venous bleeding in the depth of the wound Clamping also increases the incidence of thrombosis A serious hemorrhage may follow laceration of the saphenous vein at the femoral junction By resecting the vein 3 inches below the femoral bulb and lifting this proximal end the dissection to the bulb is made more safe The bulb should be approached from the lateral side because from the medial side the femoral vein when put on tension appears to be another branch From the lateral side, however, it is apparent that this is actually the femoral vein and not a branch

This technical point is emphasized because, at times a hemostat has been pushed through a femoral vein instead of a branch and has resulted in a large laceration in the femoral vein itself

*Laceration at the Saphenous Femoral Junction* —In case a laceration into the femoral bulb vein does occur, it is important to avoid panic. The hemorrhage at times, is terrifying

In such event pressure with a pack should be applied at the area of hemorrhage Adequate blood for replacement should be at hand or obtained at such time Skilled assistants are imperative. The saphenous vein can then be resected below lifted up and the point of laceration approached from below up A ligature then can be placed around the held-up saphenous vein and slipped up and tied above the laceration point like one does a tonsil pedicle This usually will control the bleeding If this maneuver fails it is wiser to replace a pack and hold it by pressure than to continue to try to stop the hemorrhage by clamping It is not difficult to stop the hemorrhage temporarily but it may be extremely difficult to tie off this bleeding point

Many accidents occurred due to efforts to control dangerous bleeding by ligature or clamps In the end the operator has found that he had clamped or tied the femoral artery Should the hemorrhage persist or be uncontrollable the superficial femoral vein should be resected distally and lifted up and the bleeding point approached as described under the saphenous hemorrhage Again blood replacement and excellent help must be available. In general in such hemorrhage it is best to pack such an area of

bleeding firmly and wait until twenty-four to forty-eight hours before removing the packing slowly under operating room conditions In most instances at this later time, the bleeding will be found to have stopped The danger to the patient is not the bleeding It is what is done to stop the bleeding In the serious accidents reported by Luke[15] there were 3 patients who developed gangrene after ligation and 1 serious but not fatal hemorrhage Of the 3 deaths, 1 was due to bleeding Venous bleeding can be controlled by pressure

(4) INFECTION —Local infection, thrombitis, infected ulcers, or cellulitis are contraindications to the operation, and should be corrected prior to it by local therapy and antibiotics In the clean cases, infection can be kept minimal, if there is adequate skin preparation of the operative site.

This operative site is near the labia and scrotum and soap and water preparation at the time of operation is of importance Other excretions or exfoliation of the skin then can be removed with ether and the wound can then be surgically prepared with sulfonated detergents (pHisoHex) The routine use of antibiotics without cause is injudicious In these patients, however, where multiple skin incisions are made and instruments (strippers) are passed from one point to another subcutaneously, contamination by surface or extraneous organisms can occur For this reason we use penicillin routinely unless the patient is sensitive to the antibiotic

Since the skin must be markedly undermined in removing these so-called blowouts, it is not too unusual for these wounds to heal slowly A pressure pad applied at the area will help in healing, acting as pressure does in a skin graft With the removal of the underlying veins, there is a considerable subcutaneous defect over which the skin is approximated Pressure must be maintained to keep the skin in contact with other tissue and maintain its viability

Hematomas in this area also will be slow in healing

If infection does occur, it should be drained and antibiotics used.

Cellulitis or an active phlebitis, in our opinion, is an absolute contraindication to the operative procedure We allow a minimal period of six months after clinical evidence of a thrombitis has disappeared before operating A dermatitis, particularly in the groin or subscrotal area, which cannot be adequately prepared, also is a contraindication

In certain cases where there has been an infection which has subsided, a thorough course of antibiotics prior to operation is *indicated* It is our practice, in patients who have had thrombitis now clinically subsided, to insist on a week's hospitalization with antibiotic therapy administration before the operation Occasionally, small blood transfusions may be necessary to control these infections

Avoidance of contamination of the wounds after the operation is also important The area in the groin site is especially difficult to keep adequately covered We seal these wounds as much as possible and do not disturb them for a week unless a systemic or local reaction makes it necessary to inspect them [24]

(5) POSTOPERATIVE THROMBOSIS —This complication is best prevented rather than treated The delay in operation until all inflammation of the vein or leg has subsided and remained quiescent for at least six months will

avoid this complication most of the time The occasional spread of a clot to a deep vein may occur This should be diagnosed by careful observation The diagnostic points are outlined on pages 624 to 627 In general an increased pulse abnormal pain history of previous clotting excessive swelling of the part, some cyanosis compared to the other limb, a feeling of anxiety by the patient and signs or symptoms of a small pulmonary embolism are serious signs These patients should be treated by anti thrombotic drugs to the therapeutic level Heparin can be replaced by an oral drug when the prothrombin level has been raised Bleeding can be prevented by pressure In selected instances only should a ligation of the deep veins be considered See chapter on Pathologic Venous Clotting p 607

*Certain points regarding the occurrence of accidents may be emphasized*

(1) Accidents occur in inverse relation to the skill of the operating surgeon Vein operations may be serious.

(2) If venous hemorrhage occurs it should be controlled by pressure until such time as adequate blood for transfusions and experienced assistants are at hand

Where necessary pressure alone can control sudden venous hemorrhage and a pack may be left in place If necessary at a later date the wound can be re-explored Frequently no further tying will be necessary

(3) The femoral artery is anatomically superficial in the groin and lies directly over the femoral vein

At operation time arteries go into spasm and may be mistaken for veins

(4) The best clamp in an emergency hemorrhage is the operator s fingers A pack will control venous bleeding

The clamping of vessels blindly in the presence of hemorrhage is extremely dangerous and will frequently result in complications much more serious than the hemorrhage

(5) Under no circumstances should the operation be considered without hazard especially in untrained hands

When the operation is performed it should be under operating room circumstances with a trained team and adequately trained assistants

(6) Bed rest following such an operation greatly increases the complications of thrombosis and embolism

(7) Retrograde injection definitely increases the complications of the operation

**Reasons for Recurrences** — A study of our postoperative patients made in 1946 and 1950 showed the following reasons for recurrences (approximately 20 per cent)

1 Of the recurrences 20 per cent are due to an error in diagnosis *i e* arterial varices or postthrombotic veins

2 Twenty per cent are due to technical errors in the operative technics in the groin or at the incompetent points.

3 Ten per cent are due to inadequate resection of the lesser saphenous system

4. Fifty per cent are the result of new incompetent points breaking through This latter group are unpreventable but respond to secondary resection.[31]

## PLASTIC EXCISION OF FAT PADS AND VEINS WITH VEIN OPERATION

Many of the patients with varicose veins are obese and have fat accumulation, particularly at the knee and ankle These contain large veins They are constantly traumatized by the pads on the other leg when the patient turns in bed or walks The fat becomes painful (adiposis dolorosa) and attacks of phlebitis are frequent It is our practice to resect these fat pads at the time of the operation This plastically removes the fat and veins and restores the leg to normal size It substitutes a scar for an unsightly and painful enlargement, but does not increase the magnitude of the operation to a great extent

## MANAGEMENT OF VARICOSE VEINS DURING PREGNANCY

Pregnancy precipitates varicose veins in many patients In others, it aggravates pathologic veins already present The problem is more complicated than in simple varicose veins as the back pressure necessarily raises as the pregnancy advances toward term In those with poor venous walls or valvular systems the signs and symptoms of the varicosities may be so great as to be incapacitating The incidence of varicose veins varies with the age,[32,34] frequency[1,6] and number of pregnancies and is approximately 11 per cent In the veins of the expectant mother the pressure factor is most important The venous pressure increases both in the saphenous system and in the femoral system as the fetus enlarges. The other etiological factors such as valve failure, anatomical peculiarities, etc., all play a part as do previous inflammatory venous attacks in the development of the pathology In some of these patients, the leg veins alone are involved In others, the vulva, rectal and gluteal areas all become affected. Some patients have been forced to bed during the last triad

**Treatment.**—For many years pregnancy was considered a contraindication to any active varicose vein therapy This attitude prevailed in the treatment of nearly every type of lesion including malignancy Except for very few obstetricians, most doctors in this field now believe that the developing child should in no way mitigate against necessary medical or surgical treatment It is the feeling of our Clinic that if any vascular work is required, it should be carried out as if there were no pregnancy factor. The treatment of these patients can be divided into three classes

1 *Mild, But Symptomatic Vein Problems*—In some patients only support is necessary. In the legs an Ace bandage is more effective than elastic stockings because of its adjustability An elastic girdle will give support to the upper thighs, hips and buttocks A male type of supporter which has a two-way stretch, wide elastic belt for the lower abdomen and buttocks area with a button type of scrotal support can be fitted to support the veins of the vulva Into this scrotal support can be placed cotton or sanitary pads to apply the pressure In others, the buttons can be elevated to form a more snug fit

2. *Enlarged Veins Causing Severe Symptoms*—In approximately 30 per cent simple varicose vein injections will ameliorate the symptoms suffi-

ciently to permit the pregnancy to proceed without distress. In the other half in this group the valve failure is so definite and serious that ligation and stripping of the veins is needed. The classical procedure described is carried out as if there were no pregnancy. The operation can be performed in any month of pregnancy and we have operated within a few weeks of term. A premature delivery would be considered coincidental and not as a result of the ligation procedure. To this operation must be added a resection of the veins of the vulva and at times hemorrhoidectomy, if required. As described in the chapter on Arterial Varices some changes of this type are precipitated by pregnancy.

3. *The Thrombotic Type of Veins.*—In these patients usually there has been a history of previous inflammatory or pathologic venous clotting. These patients are subject to new attacks during their pregnancy. If not inflamed these veins can be resected and stripped in the classical manner. It is certain that the incidence of the inflammatory reactions and embolisms can be decreased. This latter is important since the relative reduction in the death rate from hemorrhage, infection and toxemia has raised the incidence of pulmonary embolism to 10 to 20 per cent of the pregnancy deaths.[8,12]

It is of interest that several writers have reported on the lowered incidence of complications of pathologic veins when they are treated surgically instead of conservatively. Subsequent thrombophlebitis has been decreased 20 times in one series and the only deaths occurred in the conservatively managed patients.[14]

The patient should be informed that subsequently further therapy may be needed.

The pathologic changes resulting from a venous thrombosis and requiring medical and surgical management are described in the chapter on Pathologic Venous clotting, page 607.

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Chapter

# 31

## ARTERIAL VARICES[1,2]

**Definition.**—By arterial varices is meant pathologic enlarged veins which appear as varicose veins but which have multiple small arterial connections. These connections while of congenital origin have been closed in most instances for many years. Growth, stress, strain, pregnancy, trauma, or other variations in the living or physiologic state produce the intra-arterial rise in pressure necessary to break through the inadequate layer or layers of cells separating the arterial and venous systems. They are in fact small arteriovenous fistulas. They lack the symptoms of arteriovenous fistulas such as bruit, thrill, etc. because the arterial connections are small and the venous components large. They are the cause for pathologic enlarged veins of the recurrent type in 1 out of 6 instances.[2,3,4]

That pathologic enlarged veins develop rapidly in certain individuals has been observed by many surgeons. These veins come on suddenly and with the increase in size cause discomfort out of proportion to the time they have been present. In such cases after the usual varicose vein operation the veins promptly recur.

We have recognized that this group of patients has a different pathologic basis than the vein valve failure present in ordinary varicose veins. After years of observation it was proven that these patients have small arterial connections with their veins. This condition has been described as a separate syndrome. The pathologic picture in these patients is not caused by incompetent valves alone but is due to congenital arteriovenous connections which suddenly open and often are not diagnosed.

**Etiology.**—These arteriovenous connections are congenital in nature. The arterial and venous systems communicate prior to birth but close under ordinary circumstances during the last months of fetal life. In some where this closure is not effected congenital arteriovenous aneurysms are present. Similar but less extensive pathology may be present in the port wine blemishes, the cavernous hemangiomata and other vascular congenital anomalies. Quoting Reid, "it is a marvel not that congenital abnormal communications occur but that they do not occur more often in view of the common bed of development of each side of the vascular tree and the enormous constructive, as well as destructive, changes necessary before the final pattern is reached, that is, with formation of definitive arterial and venous channels."[4]

The closure between the artery and vein systems may be inadequate. The separation may be by *immature* cells, at times only a single layer or two of cells obliterating the openings. Such walls cannot withstand extra

pressure. At times of stress or strain, such as at puberty when there is great growth of the body or when a person takes on a type of work to which he and his vessels are unaccustomed, this weak barrier breaks down. A "blowout" occurs with the development of multiple arteriovenous connections. This abnormal connection between the two systems also may occur at the time of muscular atrophy, *i. e.*, at the time of menopause or degeneration in the viability and maturity of the tissues.

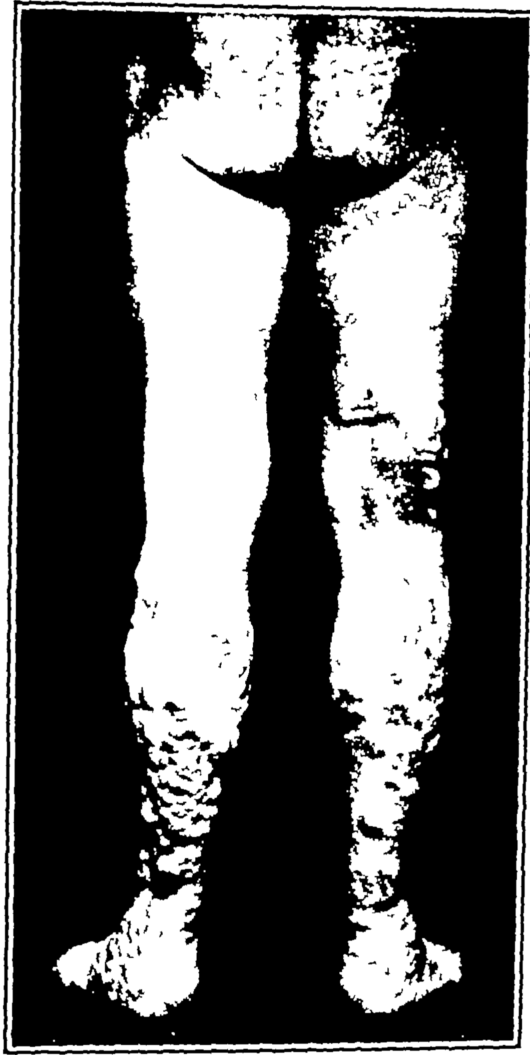


FIG. 185.—Arterial Varices. Enormous dilatations of the veins of the legs. When the veins were opened, arterial pulsation occurred. These are arterial varices and have small arterial connections which widely dilated the veins.

During World War II many such lesions arose among young men in the armed services where the unusual strain of standing and walking on their feet or the carrying of heavy loads increased the arterial pressure. We have found the lesion to occur about equally in the two sexes, although this may be a false finding because of the number of women who entered strenuous and exerting types of work during the war years.

This syndrome differs from that of the aneurysmal varix in which one small arterial branch dilates the saphenous tree. In arterial varices multiple

arteriovenous connections are suddenly opened with the increased pressure of the arterial blood rapidly dilating the saphenous system[2,3,4,5]

**Symptoms** —The most common symptom is the *very rapid development of dilated veins* which were not present before These veins fill quickly on dependency and remain partly distended on elevation of the limb The enlarged veins occur most often on the *lateral or posterior aspect of the leg* or in the popliteal space



Fig 186.—Arterial Varices Pathologic section of arterial varices Note that the blood vessel wall is thicker than a vein but does not have as many coats as an artery This is the reaction of the vessel to the arterial flow The cells are extremely immature At earlier stages the blood vessel wall may be paper thin.

There is *increased heat* in the area of communications due to the presence of arterial blood This symptom of increased heat may be the first one noticed by the patient This heat can be felt grossly with the hand and is registerable with a potentiometer The affected area is usually from 3° to 10° warmer than a similar area on another part of the body

A *bruit* and a *thrill* usually cannot be elicited The bruit probably could be heard if we had stethoscopes that were sensitive enough A bruit is caused by whirling blood the arterial blood entering a cavity or another

vessel in which the circulation of the blood has a space in which to whirl before continuing on its way In arterial varices, the arteries are small and the veins large enough so that the veins are able to take up this blood without the whirling which causes the bruit

At operation time, multiple small arteriovenous connections are seen. These, at times, appear like many venous branches, but when these vessels are observed carefully and attendant spasm permitted to relax, the *vessels* will be seen to be *pulsating*

When the vessels are incised, *arterial blood* is emitted synchronously with cardiac systole. In many instances, if a needle is introduced into one of these vessels, bright red, pulsating blood will be seen to enter the syringe The *oxygen saturation test* will show the oxygen concentration is much higher in these vessels than in other veins in the body

**Pathology.**—Gross pathology shows multiple small vessels, much like venous branches, coming off from the femoral artery and entering the saphenous tree These branches also may come from the pudendal or other branches of the femoral *artery,* to enter the saphenous system. Similar connections may arise from the popliteal artery to join the lesser saphenous system Where such connections occur, there will be secondary branches on the lateral and/or posterior portions of the leg

At operation time the multiple thin-walled vessels tear readily and their control is difficult The blood in these vessels is of higher oxygen content than venous blood

On pathologic section, the vessel walls will show greater thickness than is usual with a vein The wall is thinner than in the usual artery The more rapidly moving arterial blood causes these changes in the veins On histologic section, the actual point of anastomosis is rarely seen In most cases, the pathologic section shows innumerable vessels, not identifiable as either arteries or veins, intermixed with fat and subcutaneous tissue Most of these areas must be excised en bloc See figure 186

**Diagnosis.**—The diagnosis of arterial varices can be made on the following points (1) The veins appear suddenly and dilate rapidly

(2) These enlarged vessels occur most often on the lateral aspect of the leg or in the popliteal space

(3) There are multiple small arterial connections to these veins

(4) When such vessels are opened, arterial bleeding is present This pulsation is synchronous with cardiac systole

(5) The veins remain partially filled on elevation of the limbs The blood in these vessels can be reduced by elevation or on pressure, but the veins fill more rapidly than ordinary varicose veins on a valve failure basis

(6) If a needle is introduced into these veins, arterial blood will be found pulsating in the syringe barrel A thrill and bruit are rarely present

(7) Oxygen saturation tests carried out on the blood of these vessels will show a much higher percentage of oxygen than that in other veins in the same patient's body Oxygen saturation may be compatible with arterial blood

(8) There is an increased skin surface temperature at the point of connections. This usually is *registerable* with the hand and always with a potentiometer.

(9) These lesions are usually operated as varicose veins and in many cases have been operated previously Following the operation the veins recur promptly Reoperation in such instances shows pathology similar to that originally seen

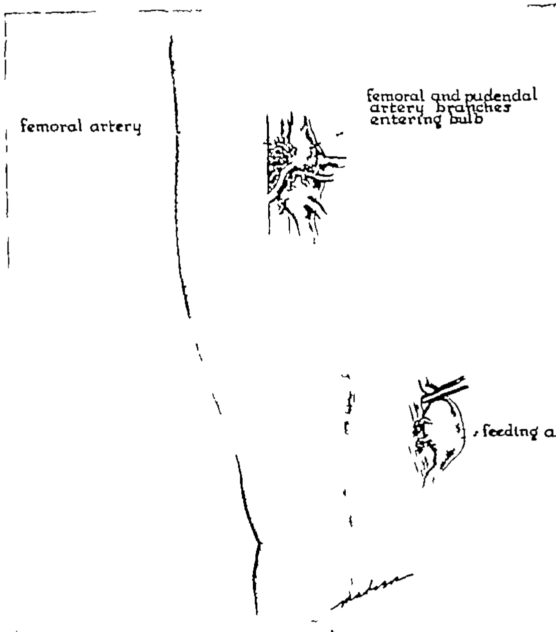



FIG 187 —Arterial varices at operation. Notice multiple small arterial connections entering the vein. Many of these are in the groin on the lateral aspect of the leg or in other instances, in the popliteal space (*Pratt, courtesy Am. J Surg.*)

**Treatment.**—The treatment of arterial varices consists of (1) Exploration in the groin at which time all arterial connections to the veins are divided and ligated

(2) Wide excision of the saphenous vein and all its branches in the groin a segment at least 4 inches in length being removed after division of all arterial and venous branches

(3) Excision of the saphenous vein at each incompetent point The test for incompetent communicating branch valves (see pages 565 to 567) indicates such incompetent points

(4) Extensive excisions of all the dilated vessels in the lateral or posterior aspects of the leg This latter often requires a block excision Generally, the skin may be undermined and all the superficial tissue excised including the blood vessels, tying each perforating vessel below the fascia The skin then is reapproximated and with pressure heals like a skin graft onto the muscle

(5) The patient must be observed at six-month intervals for perhaps five years for recurrence Recurrence is not unusual nor is it to be unexpected If there is recurrence and it is minimal, it may be handled by local injections of sclerosing solutions If the recurrence is large, it should be resected in a similar fashion to that described above Wright[7] has noted and described the frequency of these arterial varices Others more recently have reported their similar findings [8]

**Prognosis.**—The prognosis for complete cure must be guarded In many, these conditions tend to spread, new openings appearing after excision not unlike congenital arteriovenous aneurysms In this respect, the

INCIDENCE ARTERIAL VARICES IN PATIENTS WITH ADVANCED VENOUS PATHOLOGY AND IN PATIENTS PREVIOUSLY OPERATED

| | |
|---|---|
| Number of patients operated for advanced venous pathology | 596 |
| Number of patients with arterial varices | 151 (25 3%) |
| Number of patients previously operated | 361 |
| Per cent arterial varices previously operated | 41 8% |

tissue is similar to malignancy in its local spread Definite metastases are not seen although, at times, the blood vessels seem to jump to another part in a somewhat "skip" fashion The areas involved may be so extensive that surgical excision becomes impossible and it may be necessary for the part to be sacrificed In some, once the arteriovenous connections are excised, an increased load is placed on the remaining vessels and new openings occur. These are usually contiguous to the previous ones

Perhaps the strength of these blood vessels may be increased by one of the vitamin or endocrine substances This lesion warrants further investigation

One should suspect the condition (1) if the veins dilate rapidly, (2) if they develop in an individual who previously did not have enlarged veins, (3) if they occur in a young individual, (4) if the site is the lateral or posterior aspect, and (5) if they recur after apparently adequate vein surgery

Many varicose vein conditions of the extremely extensive type may have arterial connections as the underlying factor We suspect that many extensive varicose vein conditions are due to arterial connections These connections are a factor in many individuals In operating on varicose veins, one often sees the pudendal artery closely wound around the saphenous-femoral junction If this is dissected carefully, branches often

are visualized entering the saphenous tree It may be that all severe varicose veins arise from small arterial connections This point is under investigation

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# Chapter 32

## HEMANGIOMATA IN THE GENITAL TRACT. VENOUS LESIONS OF THE EXTERNAL AND INTERNAL GENITALS

THERE are many types of vascular anomalies or growths Other vascular tumors are discussed in Chapter 33 The more common ones which affect the vascular system and for which the vascular surgeon is most often consulted are included The hemangiomatous growths which affect the genital tract present a serious problem and these are discussed in detail. Enlarged veins and hemangiomata may occur in the vulva, the vagina, over the upper groin, abdomen, and in the pelvis Some of these are of arteriovenous origin and of a congenital nature Many of the birthmarks of the port-wine type and other congenital blemishes are in this category These lesions may be mild or unnoticed until precipitated by pregnancy They may progress rapidly The symptoms develop very early in pregnancy and continue increasing in severity as the fetus increases in size

**Venous Anatomy of the Pelvis.**—The vein drainage of the external genitalia follows that of the arterial supply These veins are in the form of large plexuses which intercommunicate They empty into the internal pudendal (inferior pudic) and the internal branch of the inferior gluteal vein (sciatic vein) The former vein accompanies the internal pudendal artery and empties into the hypogastric vein The latter drains into the lower part of the hypogastric vein In the *female*, the *vaginal venous supply* begins as large plexuses on either side of the vagina These anastomose widely with the hemorrhoidal plexuses, the vesical, pudendal and pampiniform plexuses as well as with the uterine plexuses Two vaginal veins, one on either side of that organ, carry the blood to the hypogastric vein The *uterine venous supply* is in the form of plexuses which are on either side and at the upper end of the uterus between the two layers of the broad ligaments They communicate with the ovarian and vaginal plexuses They drain through two uterine veins which begin low, on either side of the plexus opposite the external os They carry the blood to the hypogastric vein The *ovarian veins* develop as a plexus in the broad ligaments around the ovary and tube and are in communication with the uterine plexus These veins, like the uterine veins, dilate tremendously during pelvic congestion, pregnancy, or back pressure from a tumor The ovarian veins in this plexus are called the pampiniform plexus which corresponds to the mass of veins in the spermatic cord in the male These ovarian veins follow the

ovarian artery The right ovarian vein opens into the inferior vena cava at an acute angle The left ovarian vein passes behind the iliac colon to enter into the left renal vein at a right angle These veins have valves The variation in the drainage of the ovarian veins on the two sides has clinical importance in thrombosis or septic phlebitis of the female pelvis The *vesical plexus* surrounds the lower end of the bladder It empties into the vesical veins which vary in number They communicate with the pudendal veins These drain directly into the hypogastric veins It is apparent from this limited anatomical description that the hypogastric veins and the vessels which drain into it are the important factors in female pelvic venous pathology Other veins draining the pelvis include the superior and inferior gluteal veins the internal pudendal veins the obturator veins the lateral sacral veins and the middle hemorrhoidal veins all of which empty into the hypogastric veins

In the *male* the deep veins of the penis from the corpus cavernosum join the venae comitantes of the internal pudendal artery and drain into the hypogastric veins The *spermatic veins* in the male correspond to the ovarian veins in the female They arise as a convoluted or pampiniform plexus at the back of the testes draining them and the epididymis The pampiniform plexus makes up the mass of the spermatic cord These veins are numerous tortuous and thin walled They run in front of the vas deferens in the cord At the subcutaneous ring they become three or four veins which run in the inguinal canal to enter the abdomen through the internal ring Here they form two veins on either side These veins run on either side of the spermatic artery along the psoas muscle retroperitoneally They then unite to form one branch The spermatic vein on the right side enters the vena cava at an acute angle On the left side the spermatic vein drains into the left renal vein entering it at a right angle These anatomical variations likely cause the left testis to be lower than the right These factors may have clinical significance in thrombosis

**Symptoms** —The pathologic changes are usually in the female and rarely in the male The main symptom is a feeling of heaviness and weight wherever these large vessels appear particularly on the vulva and lower vagina There is relief upon lying down or sitting but the symptoms occur promptly when the patient stands up Such patients devise various forms of girdles suspensories or diapers to remove some of the pressure from this area when they are on their feet In extreme cases some of them have to spend most of the time in bed These symptoms are aggravated during pregnancy or in the presence of tumor masses or other causes for pressure or congestion The external veins may dilate and appear like clusters of grapes. They protrude and at times rupture with slight trauma The entire vaginal canal may be deep blue in color When dilated with a speculum the mucosal and submucosal layers are seen to be filled with distended veins Usually the veins of the legs become dilated the symptoms varying from those of simple varicose veins to those described under Arterial Varices.

**Pathology** —The pathologic changes are due to obstruction of the involved veins and valve failures and vary with the degree and site of the obstruction (see Anatomy on page 596) Thrombosis may occur This complication and embolism are discussed under pathologic venous clotting

In some patients, the vulva is like a sponge when distended and is composed mainly of skin, mucous membrane, and dilated vessels under the coverings. The vessels swell enormously when the patient is on her feet. On section, large vascular sinuses are seen, usually valveless. The vessels are immature. In some, arteries and veins communicate. Arteries and veins *per se* often cannot be identified one from the other. The pathologic changes in those with enlarged veins in the lower extremities already have been detailed.

**Treatment.**—The need for treatment often arises during pregnancy. Treatment depends upon the severity of the symptoms. In the mild cases, mere support is sufficient. In those seen during pregnancy, some type of athletic supporter of the male type, with an elastic portion between the legs, may give temporary relief. While some of the symptoms may subside with delivery, the underlying pathology remains and will reappear spontaneously or when the excitement (pregnancy) again is a factor.

Many times conservative management will be impossible and the distress and discomfort of the patient requires surgical intervention.

*Operative Treatment.*—If the condition is extensive and involves the pelvic veins, the operation will be similar in the pelvis to that performed for enlarged veins in the leg. The pelvis must be opened and the extent of the pathology determined. In most cases, the hypogastric vein must be ligated at its junction to the common iliac vein. In others, resection of the pampiniform plexus in the broad ligament has been sufficient to control the situation. This usually should be accompanied by resection of any pathology found in such organs. The dissection and resection of the ovarian veins may be necessary as in the septic thrombosis discussed in that chapter. In some patients, hysterectomy has been required to resolve the process. Where simple varicosities of the broad ligament exist, they should always be excised and not merely ligated.

*Resection of Veins of the External Genitalia.*—These veins can be locally resected after careful preparation of the operation site. It is necessary, in addition, to resect the pudendal branch of the saphenous vein. These local resections are successful and alleviate the symptoms.

*Sclerosing Injections.*—The remaining veins after resection can be sclerosed. The injections should be done carefully with a minute fine-gauge needle, being certain one is in the vein lumen. The difficulty of applying pressure in such an area adds to the complication of these injections. Manual pressure should be applied until the injection aperture has been sealed. A T-bandage combined with a vaginal tampon may be effective.

*Cryotherapy of Freezing.*—Carbon dioxide ice has been used in some hemangiomata. It is held with pressure against the area for approximately fifteen seconds, and repeated every two weeks for from 2 to 8 applications. This ice is supposed to injure the endothelium with thrombosis which is followed by organization. Such therapy has not been too successful.

*Radiation Therapy.*—These tumors do not respond well to irradiation. Radiation has been tried in the younger age group. Unfortunately, normal tissues, particularly bones, also are sensitive. The unpredictable after-results which include telangiectasia, scarring, alopecia, necrosis and carcinoma make such therapy inadvisable.[1-5]

**Pathologic Venous Clotting in Veins of the Genital Tract** —Such changes may be primarily inflammatory or predominantly thrombotic. The lesions may be mild or spread rapidly or become suppurative. Embolisms are not infrequent. The diagnosis and treatment of such lesions are considered under the chapter on Pathologic Venous Clotting (see pages 607 to 650)

**The Management of Varicose Veins During Pregnancy** —The treatment of ordinary varicose veins during pregnancy has been discussed on pages 586 to 587. The pregnancy status does not interfere with the treatment of such lesions.

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# Chapter 33

# TUMORS OF BLOOD VESSELS, BENIGN AND MALIGNANT

TUMORS of blood vessels can be divided into two types, the benign and the malignant The hemangioma is a classic example of the benign type and is the most common tumor of infancy and childhood A classification from the tumor standpoint has been made[14] A modification of such classification from the vascular standpoint may be useful Some of these tumors are arteriovenous aneurysms and thus the classification and discussion of the lesions overlap Many of these vascular masses, while microscopically benign, actually are malignant in that their complete excision is impossible By local extension they may involve structures the excision of which surgically is impossible

(See Tumors of Heart, and Tumors of the Pericardium, pages 143 and 155 )

## CLASSIFICATION

### I Benign Blood Vessel Tumors

1 SIMPLE HEMANGIOMA —(*a*) Capillary type these vary from a simple dilatation of the capillaries to the port-wine stains (nevus vinosus) and the organizing or sclerosing hemangioma

(*b*). Cavernous hemangioma These may be on the skin or in the viscera

(*c*) Cirsoid hemangioma Most of these tumors are of arteriovenous origin Virchow described this tumor as similar to a "pulsating mass of earthworms "[22]

(*d*) Systemic hemangioma These are very extensive and diffuse tumors which may cover half of the body's surface or half of the head or trunk As a result of the increased blood supply the tissues and bones become enlarged Actually, these are congenital arteriovenous aneurysms[11 14–19] The extent of the arteriovenous shunt depends upon the number of connections

(*e*) Hypertrophic hemangioma This is a solid, purplish-red tumor. These tumors never regress spontaneously and the tumors are resistant to both sclerosing solutions and radiation. The endothelial cells are the proliferative units

2 CONGENITAL TELANGIECTASIA (Rendu-Osler-Weber's Disease)[23] These hereditary lesions affect and are transmitted by both males and

females They may skip generations[9] These tumors appear later in life (the fourth decade) but nosebleeds in children may herald their future onset There are no petechial or ecchymotic manifestations They must be differentiated from hemophilia pseudohemophilia and thrombocytopenia purpura





Fig. 188 Fig 189

Fig 188.—Congenital hemangioma and arteriovenous fistula involving the right lower extremity genitalia bladder and all pelvic organs including the bowel. Multiple resections resulted in recurrences Deep tissues including the bone had been invaded. Autopsy showed invasion similar to malignant infiltration. Surgical eradication or X-ray therapy ineffective

Fig 189 —Cavernous hemangioma in girl age six. Note hypertrophy of part and involvement of the skin Standing produced so much blood loss to the extremity that syncope followed. Child had never walked prior to therapy Surgical excision successful

3 Neurocutaneous Angiomata —In this group will be the angiomas of the skin which are associated with von Recklinghausen s neurofibromas The brain lesions sometimes called Pringle s Disease and Bournesville s Syndrome which are tuberous sclerosis and those lesions involving the face and brain unilaterally adequately described by Cushing[7] in 1903 but bearing the name of Stürge-Weber s Disease, fit in this group



Fig 190 —X-ray of patient in figure 189 indicates large vascular tumors and blood sinus spaces



Fig 191 —Surgical treatment of patient in figure 189 Skin removed at [illegible] of an inch thick with electric dermatome All superficial and deep tissues including tumors and the deep fascia excised Epithelium replaced after discarding diseased part

4. VISCERAL HEMANGIOMA —(a) These may occur in the eye and the form described by Landau and von Hippel[15a] is hereditary. Cysts as well as hemangiomas may involve the cerebrum, the cerebellum, the medulla and the spinal cord.

(b) The tongue is often a site for hemangiomas. The tumor may interfere with eating and a slight injury may result in severe hemorrhage.

(c) Gastrointestinal tract. The small intestine and colon are the most common sites of these tumors. The stomach rarely is involved. In unexplained intestinal bleeding they should be considered.[4]

(d) Liver. Most of the hemangiomas of the liver are found at autopsy. Occasionally symptoms may develop. These tumors are of the cavernous hemangioma type. Resection has been the most effective treatment.[24]



Fig 192.—Superficial and deep fascia blood tumors and the rest of the epithelial layers of patient in figure 189. Note invasion of deep tissue.

(e) Muscle. One or more muscle groups may develop tumors of a hemangiomatous type. The most common site is the forearm and the muscle most often involved is the triceps.[8]

(f) Bone. The skull is most often involved. The vertebræ, especially the lumbar, the scapula, ribs, fingers and pelvis may be affected.

II **Malignant Blood Vessel Tumors**

1 ANGIOSARCOMA —This lesion relatively is rare. Only 20 cases were collected out of 56 malignant blood vessel tumors in a ten year survey at Memorial Hospital (New York).[13] This number is contrasted with 1 050 benign angiomata in the same period.[14]

*Etiology* —Fourteen of the 20 patients reported were of the Jewish race. There is no sexual differentiation. Most of these patients were under forty

years of age Over half of these lesions were on the extremities The malignancy is considered to develop from the granulation tissue capillaries in traumatized areas with the gradual development of angiosarcoma

*Pathology* —The *gross* pathology resembles that of Kaposi's sarcoma The lesion is a firm, large tumor deep in the soft tissues, surrounding bone

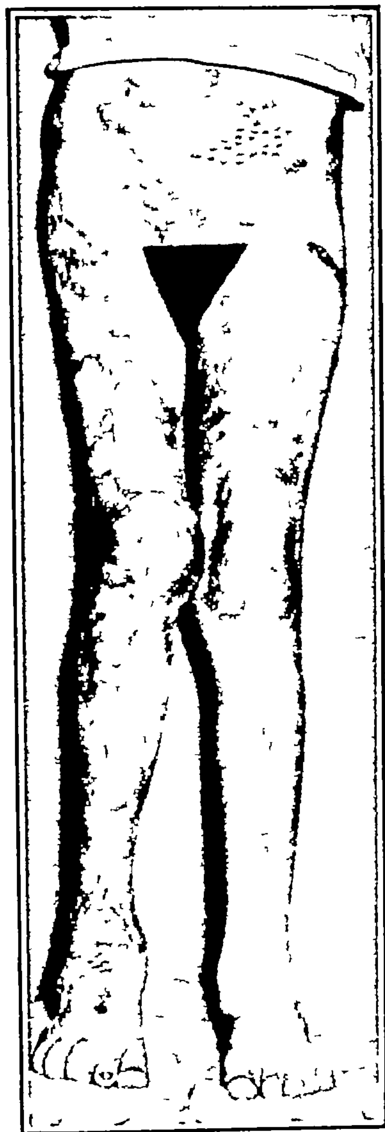


Fig 193



Fig 194

Fig 193 —End result patient figure 189 Epithelium obtained from opposite limb and abdomen Patient able to walk Patient referred by Dr B Milanes Lopez of Havana, Cuba

Fig 194 —Congenital hemangioma complicated by arteriovenous fistula in patient aged twenty-one Treated by excision and electric dermatome graft End result cure (Three years )

or tendon and invading muscles, fat and veins With the rapid growth there is hemorrhagic cyst formation, necrosis and degeneration Pressure causes symptoms of pain and necrosis *Microscopically*, the sections vary. Comparative biopsies and silver reticulin stains are necessary Often, cavernous sinuses are found The cells may be rounded, fusiform or polygonal

*Symptoms*.—The tumor develops most often on the extremities, grows rapidly and is bulky. It causes pain and edema. Other symptoms depend entirely upon the site of the lesion. Metastases to the lungs, other viscera, bones and lymph nodes are common. Death occurs in approximately three years. A second common site of the tumor is the nasal cavity or the sinuses. The lesion has occurred in the breast.

*Treatment*.—Radical amputation is the treatment of choice in angiosarcoma of the extremities. X-ray and radium is indicated for the lesions in the nose and in the paranasal areas. It has been curative in some and palliative in others. Only 17 per cent survive three years, 9 per cent for five years. The average survival rate is two and a half years.[11]

2. KAPOSI'S SARCOMA.—This lesion has been called an idiopathic multiple hemorrhagic sarcoma. Since its description in 1872 by Kaposi,[12] 600 patients have been correctly diagnosed. It is probable that many more patients have been misdiagnosed.

*Etiology*.—This lesion occurs most often in the extremities (87 per cent) of patients over forty (78 per cent). It affects the Jewish and Italian males in Russia, Italy and Poland.[13] The preponderance in males is striking (95 per cent). It is considered likely that there is some systemic carcinogen which acts specifically on the vascular tissues.

*Symptoms*.—The sarcoma originates usually in the skin. A red demarcated macule increases in size. There is usually some edema. As the lesion grows and becomes elevated, a blue-red color appears denoting the angioma. It may look like a melanoma. The tumor multiplies, becomes less vascular and attains the indurated sarcoma state with elevated bluish-black appearance. The area frequently involves similar parts of the other extremity. Edema results and the tumors may ulcerate or hemorrhage. Pain is associated only with those on the bottom of the feet, or on the penis. With further growth lymphadenopathy develops. Terminally, the tumor may be in the submucosal layers of the gastrointestinal tract or lungs. Fatal hemorrhage may result. The oral cavities and bone have been invaded. The tumor has been noted in the heart,[14] kidney, liver and intestines,[21] lymph nodes, penis,[7] eye,[10] nose and ears.[28]

*Pathology*.—The *gross* pathology shows an inflammatory like macule. This develops into a granulomatous, bluish red nodule like an angioma. It progresses to the black plaque-like appearance of sarcoma. *Microscopically*, in the early macule stage, the skin vessels are connective tissue proliferations. Endothelial cells form vascular sinuses similar to hemangiomas. This is an abnormal formation and red cells escape into the tissues with hemorrhage. In the final stage, there are spindle cells typical of sarcoma with some hemangiomas. Old blood pigment is present. These marked variations in the histologic picture are common. Some tumors have large collections of cells of a spindle or endothelial type and simulate melanoma, angiosarcoma or endothelioma.

*Treatment*.—Surgery. Excision has yielded the best results. In widespread multiple lesions, such treatment is not possible.

Radiation. Radiation has been used extensively. The tumor is radiosensitive. The early lesions will regress satisfactorily. If the tumor is extensive and in the presence of edema, only mild X-ray treatment is advo-

cated Since the blood supply is not too satisfactory, overtreatment results in necrosis, gangrene and secondary infection

3 ENDOTHELIOMA —These malignant type of tumors resemble both carcinoma and sarcoma They arise from the pleura, the peritoneum, the dura, the lymphatics, the liver, brain, skin, and the testicle, ovary and parotid gland *Grossly* the masses are grey and nodular with areas of necrosis and ulceration Microscopically, the picture is typical The columnar and plexiform arrangement of cells often corresponds with the course of blood vessels Hemorrhage into them occurs at times They infiltrate widely and rapidly, and metastasize through both the lymphatics and blood vessels Their surgical excision or irradiation rarely is effective

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Chapter

34

# PATHOLOGIC VENOUS CLOTTING

*Mechanism of Venous Clotting, Thrombitis Suppurative Thrombitis, Intracranial Thrombosis in the Puerperium Venous Thrombosis Effort Thrombosis Obstruction of the Superior Vena Cava*

**Terminology** —The problem of pathologic venous clotting has become so serious that its treatment necessarily must be one of prime interest to all physicians and surgeons This includes those who are concerned with the problem primarily the obstetricians with postpartum cases the surgeons with their postoperative thrombotic patients and the general physician who has this complication as a secondary factor in the treatment of some other initial lesion One of the greatest problems is definition of the terms used to describe this clotting In one state the clotting occurs primarily and the inflammatory changes are latent and of secondary importance Many terms have been used to describe this condition Phlebothrombosis bland silent innocuous and secret all have been utilized to describe this entity In general this lesion is a soft acute clot arising secondary to an operation a postpartum delivery trauma or after some disease which requires bed rest The lesion often is not diagnosed and pulmonary embolism may result with pneumonia or even death The ratio between such clots in medical or nonsurgical patients and those after operation is approximately 8 to 1 [4]

To simplify the classification of these lesions the ones which are primarily due to clots and only become inflamed secondarily are called *thromboses*

Other patients develop an inflammatory change in their veins The symptoms in this group are those of redness heat, pain streaking leukocytosis and at times chills and fever To separate these lesions from those that primarily clot we designate all of these as *thrombitis* In these the inflammation initiates the process and such clotting as occurs results from the stasis and clotting changes secondary to these manifestations About 1 per cent of the postoperative or postpartum patients develop one or the other types of pathologic clotting Seldom has there been such an extreme degree of difference of opinion as exists at present in the management of these conditions If this problem is difficult for those who are studying these patients all the time it must be nearly insurmountable for the physician or surgeon who sees the occasional case

The investigator who reports a new type of therapy must accept a responsibility If his first enthusiastic report is justified by subsequent follow up he should so declare it If he must qualify or reverse his initial

conclusions, he should be honor bound to do so, with due reference to the previous articles. It takes approximately two years for any new therapy to be adopted generally If the original impressions change, this variation should be reported within that period of time Too often, a "hallowed" report is accepted as truth by the general medical public because of the qualified medical source Follow-up work may demonstrate that such original suggestions were not infallible If these findings are not corrected, the medical therapist may utilize them to the detriment of the one for whom we are all working, the patient This is true, particularly, in the problem of pathologic venous clotting Where it has been necessary to report 286 different articles on this subject in one year,[73] it is certain that the final treatment in such therapy has not been determined

**Historical Background.**—It has been nearly one hundred years since Virchow[83] established the fact that most clots in the lung originated from one or more emboli from another part of the body He further demonstrated that thrombosis could occur in blood vessel walls without locally inflaming them, provided the clot did not remain stationary for too long a time The appearance of these thrombi was described after being macroscopically and microscopically examined by Kahn[41] in 1890 In 1895, Aschoff[3] proved quite conclusively that pulmonary emboli, especially the fatal ones, most often arose in the large vessels in the legs or pelvis It was his belief that these clots originated most frequently in the femoral vein, sometimes in the iliac vein, or occasionally in the pelvic veins He stated that these clots propagated until they reached sufficient size to close off the pulmonary artery It is apparent that only in vessels of this size can emboli of sufficient magnitude to close off the pulmonary artery arise

It has been in the last twenty years, however, that it has become increasingly evident that most of these clots originate, at first, not in the large vessels, but have their onset in the smaller peripheral veins of the leg and then propagate proximally The work of Binschwager[10] and Roessle[71c] in Germany, of Hunter and his associates[37] in the United States, and of Bauer[9] in Sweden confirmed these theories The common origin site has been further located as in the calf vessels, particularly the posterior ones most subject to pressure and trauma (See Venous Anatomy, pages 403 to 404 )

For at least fifty years, therefore, we have had most of the knowledge we have today concerning the source of pathologic venous clotting and pulmonary embolism

## THE MECHANISM (PATHOGENESIS) OF VENOUS CLOTTING

As surgeons, we are vitally concerned with the normal functioning of the clotting mechanism Without it, operations, uniformly, would be fatal As Wright[88] has said, man throughout his entire existence is almost constantly hemorrhaging and thrombosing Normally, there is a perfectly balanced mechanism which prevents either the tendency to bleed or to clot from becoming overactive. Our studies of this mechanism and its control still are elementary Our thoughts are influenced by study in this field in the last twenty years and close observation of over 10,000 general and vascular surgical operations In additic[illegible] e closely [illegible] led

observation has been made upon an average of 5 patients with pathologic venous clotting per hospital days for seven years a total of over 14 000 patient hospital days Specifically we have studied the effect of the antibiotics anticoagulants blood transfusions and surgical operations on the blood count the platelets the bleeding and prothrombin time the protein fractions and the amount of calcium in the blood [illegible] In 1949 according to the accumulative index there were 200 articles written on this subject In 1951 there were over 650 articles written on the same subject It is certain that if so many articles must be written we do not know the final answer to the question One or two of every 100 should not die of a clot.

**Biochemical Factors in Blood Clotting** [illegible]—Perhaps if we could answer Best s question why blood remains fluid we would be closer to the answer to our problem [illegible] We know that under certain conditions such as jaundice anemia liver disease avitaminosis etc *bleeding tendencies* exist Certain groups of people such as hemophiliacs bleed abnormally In other disease states such as polycythemia and malignancy there is an increased clotting tendency This occurs physiologically also as with obesity increased cholesterol diet and the changes following venous stasis We now accept the fact that clotters exist the same as we have known for a long time that bleeders are present in the population The familial and environmental status plays a part The study of the effect of diet age activity nicotine and the status of the sympathetic nervous system barely has begun We know that most killer clots originate at other than the operative area. It has been proved also that we cannot ligate all of the veins in the body between the operative site and the lung to prevent an embolism This problem increases in importance as we operate on older patients with diseases previously considered incurable as for example malignancy

The early work of Morawitz[45] and Howell[34] and other physiologists on blood clotting brought out the classical theory of blood clotting This theory required the presence of four substances to cause blood to clot (1) thromboplastin (2) calcium and (3) prothrombin which together formed thrombin and (4) fibrinogen which provided fibrin

In the early 1940 s Quick[71a] found that prothrombin actually was two substances One of these is prothrombin which decreases when vitamin K depletion in the body occurs The other was called Factor A or labile factor by Quick Factor V by Owren[61] and Ac-globulin by Seeger[72a] The body rarely is deficient in it. The exact method or mode of these clotters actions is unknown but the classical blood clot theory so long utilized must be changed

The action of thromboplastin is little known It becomes active when a platelet factor acts with a plasma constituent called by Quick a thromboplastinogen

A chain reaction appears to guide the five clotting agents but a decrease in any one may cause bleeding

For the lysis of platelets thrombin is required As these cells liberate their clotting components more thromboplastin is formed which causes more thrombin to be formed This thrombin then causes more lysis of platelets The control of the reaction is fibrin which absorbs the thrombin rapidly

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**Biochemical Factors in Blood Clotting**[44]—Perhaps if we could answer Best s question why blood remains fluid we would be closer to the answer to our problem[11] We know that under certain conditions such as jaundice anemia liver disease avitaminosis etc *bleeding tendencies* exist Certain groups of people such as hemophiliacs bleed abnormally In other disease states such as polycythemia and malignancy there is an increased clotting tendency This occurs physiologically also as with obesity increased cholesterol diet and the changes following venous stasis We now accept the fact that clotters exist the same as we have known for a long time that bleeders are present in the population The familial and environmental status plays a part The study of the effect of diet age activity nicotine and the status of the sympathetic nervous system barely has begun We know that most killer clots originate at other than the operative area It has been proved also that we cannot ligate all of the veins in the body between the operative site and the lung to prevent an embolism This problem increases in importance as we operate on older patients with diseases previously considered incurable as for example malignancy

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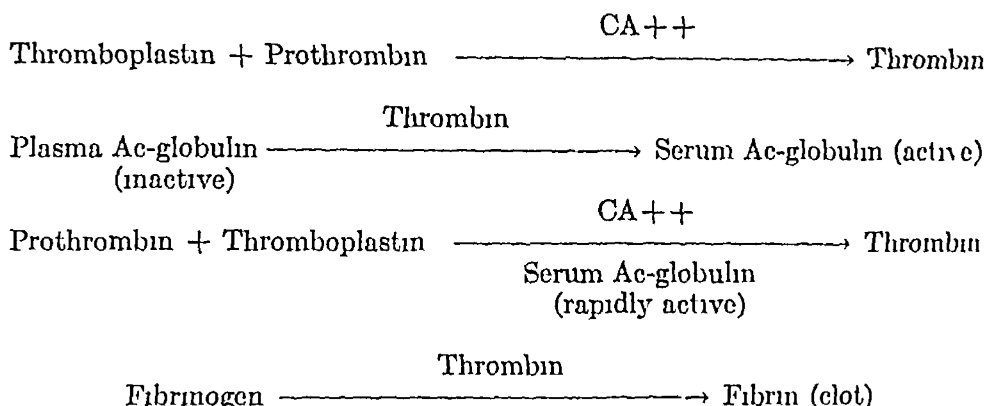
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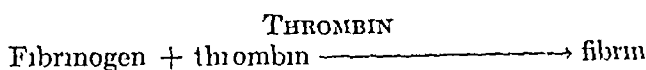
Calcium is necessary for clotting but can be discounted, as the necessary amount is always present in the body except in tetany Thrombin activates fibrinogen The final fibrin is formed by the conversion of fibrinogen The deeper one delves into the chemical theory of clotting the more complex becomes the problem It becomes apparent that Aschoff well summar-

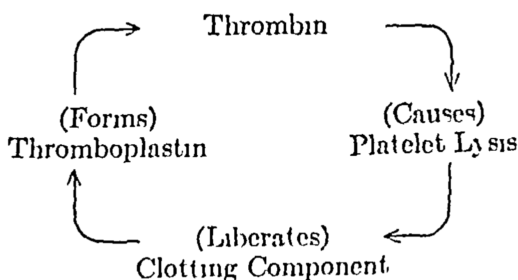



ized the clotting problem when he declared that it was "a function of a number of variables"[3] Thus, a perusal of what is known biologically and chemically of clotting leaves many links to be filled in the chain that starts with prothrombin and ends in fibrin clot

Quick's formula [71b]

Thromboplastin complex (which is thromboplastinogen + platelet factor)
+
Prothrombin complex (Ca + labile factor [called Factor V or Ac-globulin] + prothrombin)




Fibrin clot controls reaction by absorbing thrombin

**Physical-Chemical Factors in Blood Clotting.**—The physical-chemical side of the clotting problem is important Blood will remain in a fluid state for a long time if it is undisturbed and its container intact Thus, the blood in an isolated blood vessel does not clot for some time if the endothelial lining is in continuity We know that blood clots more rapidly if the blood is at *body temperature* Other factors favor clotting: (1) raised temperature—warming tends to increase clotting, (2) certain biological substances—if certain biological substances, *i e*, *poisons* such as snake

venoms are added the blood will clot faster (3) foreign surface—if the blood is on a *foreign surface*, clotting is more rapid (4) irritation or agitation—if the tissues containing blood are injured or agitated the clot forms rapidly

Certain factors favor non-clotting (1) endothelial surface intact—if there is a normal *undamaged* endothelial surface of the blood vessel there is less clotting (2) lower temperature—if the *temperature* of the part is lowered the clot tendency decreases (3) coated surface—if the blood container is *coated* with paraffin or collodin clotting is retarded (4) certain salts—the addition of certain *salts* such as sodium chloride will reduce the clotting tendency (5) tissue extracts—*tissue extracts* such as heparin will keep the blood in a fluid state for an indefinite time

A physical force keeps the fibrinogen molecules and other blood components apart and prevents their adhesion to each other and to the vessel wall There is an electric reaction between a solid surface and its fluid content This develops by selective absorption The electrical charge of both the blood constituents and their container theoretically is important Substances with the same electric charge are repulsive to each other The positive ions are attracted to the wall. A physical substance called the *zeta potential* becomes important The zeta potential is the difference between the wall potential and the center of the solution which has been termed zero The zeta potential is measured by producing a streaming potential as when blood is poured through a tube or capillary [illegible] Heparin and other anticoagulants increase the negative charge or zeta potential Knisely [illegible] photographing the process of blood sludging proved pictorially what has been known for one hundred years [13] Blood flows in a laminated fashion The blood cells are in the center of the stream surrounded by layers of plasma The central cells move the most rapidly The platelets being the smallest and the lightest are on the outside They are not in contact normally with the wall but with any type of injury they immediately adhere due to fibrin Leukocytes become attached and the red and white blood cells which normally tend to repel each other, then join the mass with a resultant propagation of the clot They do not adhere to a normal intact vessel wall

The Adherence of Platelets —The number of platelets increases to between 2 to 3 times the normal postoperatively and in the postpartum period [73, 87] These substances also become adherent between the sixth and tenth day [88] There is likewise an increased incidence of thrombosis at these same times In certain diseases platelets become covered with a film of fibrin Perhaps this film overcomes the electric repulsion charge The adhesiveness varies in different individuals at various times and under certain physiologic and disease states The degree with which thrombosis is seen to occur in the arteries or veins of certain patients after a break from not smoking would indicate that nicotine affects the adhesiveness of the platelets or cells or both [31, 73a] Occlusion in small vessels can be produced by platelets alone and begins in ten seconds after the injury and may be hemostatic in four minutes [2, 31, 73, 83, 84] Anticoagulants appear to reduce this adhesiveness The part that hydrophilic colloids play is uncertain These substances are raised quantitatively after an operation and reduce the electric charge of the cells Such a condition tends to a greater cellular

agglutination[45] The rate of flow in a large vessel is rapid and this rate decreases so that in the capillary bed its motion is minimal An injury, therefore, affects the sludging more rapidly at the capillary level Knisely's[44] photographs demonstrated that with stasis, trauma, or certain disease states, such as malaria, the cells settle on the lower side of the vessel When these cell masses slow down below a "critical speed," they may settle out as masses That such settling will occur more rapidly in injured vessels, or where the amount of fibrin is greatest, is logical The reverse, also, is true in that if the repulsive electrical charges are great, or there are anticoagulant factors in the blood normally, due to disease states or to therapy, there will be less clotting and propagation of clot Again, as in biochemistry, only the surface of the physicochemical possibilities of blood clotting has been lifted for our inspection

## BASIC CLINICAL FACTORS IN ABNORMAL CLOTTING

If we summarize these factors which we have discussed, it is apparent that any change in the relative cellular and fluid content of the blood can effect thrombosis Thus, an actual increase in the cells (polycythemia) or a relative elevation (hemorrhage) are stimulators to clot formation Hyperglobulinemia and hyperproteinemia cause more clotting A change in the electric charge of the cellular elements plays a part An increase in the number or the intangible "adhesiveness" of platelets and other cellular elements is important Age, obesity, as well as disease states, effect clotting The postoperative status, with the alteration required by starvation, blood loss, depletion of the enteric content by enemas, diarrhea or enterostomy, alters the physiology and, if stasis is added, we have the factors necessary for pathologic venous clotting We emerge from such discussion with three basic physical and biochemical points necessary for thrombosis A fourth one is included In order of importance from the surgical standpoint, these are:

(A) *Change in the Clotting Tendency.*—The first requirement for a pathologic clot is the one nebulously described as a change in the clotting tendency. It is apparent that we cannot label correctly or distinctly this component This is our unknown It probably controls itself if trauma and stasis are minimal

(B) *Injury to the Blood Vessel and Its Endothelial Lining*—This injury is inevitable in a surgical or obstetrical operation It occurs, likewise, in manipulations such as fracture reductions Pressure on the walls of vessels occurring during bed rest alone can disrupt the endothelium and instigate clot Even a slight break in the continuity of the lining cells of the blood vessels may initiate the clot formation

(C) *Slowing of the Blood Stream*—A reduction in the rate of blood flow at points in the circulation is usual with a surgical procedure Anesthesia, depressing drugs, long periods in one position on the operating table, and *bed rest*, contribute to the stasis. The necessary cutting, hemostats, ligation and retraction are added causes Stagnation of the blood, alone, can propagate clot These two factors, trauma and stasis, are interrelated as to cause and effect, Either one may result in the other.

(D) *Inheritance* —A fourth factor is the thrombotic inheritance in certain families or individuals

EFFECT OF OPERATION OR PATHOLOGIC CLOTS ON LABORATORY TESTS[47]—There have been contradictory or equivocal reports of the effects of various therapeutic measures on the components of blood It had been our impression clinically that what changes occurred due to an operation blood transfusion and the antibiotic or antithrombotic drugs etc in general were not distinguishable with the tests now available Blood counts bleeding times sedimentation rates the component proteins, the prothrombin times and the platelet counts therefore were tested to prove this point Our patients were divided into two groups those undergoing general surgical procedures and those who were being treated for active thrombosis These laboratory measures were performed the day before operation, the first the third and the seventh postoperative days. The same tests also, were done before and after the institution of antibiotic or antithrombotic therapy While it is difficult to draw conclusions from any such study certain points or patterns developed These can be summarized as follows

1 A surgical operation does not affect materially the hemoglobin or blood cell components or their differential count unless there is hemorrhage blood replacement, or infection

2 The bleeding and coagulation time is lowered slightly on the first to third day after operation but returns to normal within a week.

3 The prothrombin time is increased with blood transfusion and decreased with operation consistently but only slightly unless the operation is excessively long or the transfusions excessive

4 There is no detectable change in the albumin or globulin ratio or the total protein content.

5 The platelets consistently double in number the day after operation but are back to normal within seven days The changes in their agglutin ability have been noted

6 The antibiotic drugs reduce the expected leukocytosis and raise the prothrombin time but do not affect other chemical changes

7 The injectable antithrombotic drugs prolong bleeding time and affect the prothrombin time but little The reverse is true with the oral drugs The effect in general is quantitative but varies with age weight and disease states

Since we know definitely that operation blood transfusion and the antibiotic drugs have an effect on clotting we must accept the fact that our tests are not sufficiently sensitive to determine these changes This again points to the fact that the clotting mechanism is a delicately poised one and that a minute fractional change at least in some individuals is all that is necessary to overturn its proper function

These findings are in line with those reported by Friedman *et al* [38] who used clotting tests on normal individuals and on patients in diseased states Using bleeding heparin retardation protamine-heparin titration range prothrombin and platelet counts he found a wide range of variation but most within so-called normal limits It may be well to reconsider what is a normal value and narrow its confines

Therefore, the clotting factor is an indefinite cover-all group and clotting tendencies cannot be determined by present laboratory methods.

There are predisposing conditions, however, which would lead to abnormal clotting

## FACTORS AFFECTING NORMAL CLOTTING

**Patients in Whom Abnormal Clotting May Be Expected.**—1. *Cardiovascular Disease* —Patients with rheumatic heart disease and, particularly, those with auricular fibrillation have increased clotting tendencies This group of clotters terminate fatally from thromboembolic episodes in one out of three instances In 100 fatal pulmonary embolism patients, 50 per cent had antecedent cardiovascular disease [70] In commissurotomy operations for mitral stenosis, clots are found in the auricle in 20 per cent (Bailey reported 25 per cent )[5]

The group with coronary and cerebral thrombosis require special attention Their thrombotic tendencies specifically involve critical vessels which, if they clot, result in fatality.

2 *Malignancy and Other Degenerating Diseases* —The co-occurrence of pathologic clotting and malignancy is established Where no other cause for an inflammatory vein clot can be found, malignancy must be ruled out Trousseau mentioned the coexistence of the two diseases one hundred years ago [81] Its importance is reflected by our experience in finding 5 cancers of the uterus in one year in patients referred for an evident lesion in the veins of the legs It is significant that not one of these patients had had a pelvic examination and that each resisted the suggestion of its necessity. The frequency with which phlebitis complicates carcinoma of the pancreas is established [74, 80, 81] With operation in more advanced malignancy, the clotting problem will be more common and will be prognostically and diagnostically important at an earlier date The cause for the thrombotic tendency probably is a release of clotting fractions with tissue destruction The failure of the anticoagulant drugs to be as effective in the presence of carcinoma is established and often diagnostic and explainable in that the tissue initiates clot on a foreign body basis Other degenerative diseases predispose to clotting

3 *Age* —Increasing age brings more clotting as it does all other complications In reviewing 100 fatal embolisms, 95 per cent were over 45 years of age [70]

4 *Surgical or obstetrical operations* predispose to pathologic clots

5 *Obesity.*—The obese patient has a definitely increased clotting potentiality This can be demonstrated with a high cream diet in the experimental animal There are twice as many postoperative thrombotic incidences in patients who weigh 200 pounds or over than in those under that critical weight [73b] The lipid content, therefore, is important in clot formation, as is the type of cell, *i e* , the large, fat molecules described by Gofman [28]

6 *Fungus and Allied Infections* —The fact that fungus infection is present in most patients who have clotting episodes has been noted The possibility that such infections only break the skin to permit the acces-

of other organisms has not been evaluated clearly The circulating "tid" form may be the cause Perhaps the complication occurs due to the diseased blood vessel status

7 *Nicotine* —Smoking probably predisposes to clot formation This may be due to spasm of the vessel or an adrenalin or pituitrin like reaction within it

8. *Familial Clotting* —There are many examples of familial clotting One of our patients on whom 4 sympathectomies and a vena cava ligation for a migrating phlebitis and multiple embolisms have been performed has a twenty-one-year-old daughter This young woman had phlebitis with four pregnancies one pulmonary embolism and recently survived the resection of 4 feet of small intestine from a venous mesenteric thrombosis Another patient the wife of a surgeon demanded a vein ligation during her ninth month of pregnancy She believed that she was developing a clot as had other members of her family during pregnancies Despite equivocal symptoms, she was operated and a large soft clot was removed from her femoral vein of a size sufficiently large to have occluded her pulmonary artery and to have caused death

Wright s report of 8 members of one family with phlebitis or embolic phenomena testifies to the frequent familial tendency [16]

9 *Patients with Pathologic Veins and Previous Clotting History* —Chronic recurring thrombophlebitis is an increasingly common disease Such patients if subjected to operation may be expected to have a thrombotic complication To this group can be added the patients with severe or recurrent venous pathology *i e* varicose veins or ulcers These require special consideration and treatment

**Clotting and Cold** —That cold has a specific and predictable effect on blood seems established This is not the effect of temperature change alone There are cold proteins which can be precipitated These produce agglutinins The critical temperature of cold which produces clotting is approximately 37° F Reduction of the body temperature to this level occurs normally only in the extremities If the part is not warmed within a reasonable time or is subjected to trauma at the same time severe clotting or destruction of tissue results Recent examples were the frostbites which followed exposure of the First Marine Division at the Chanjin Reservoir in Korea Motion of the part and release of pressure reduces the necrotic incidence. The trauma may be only pressure as was indicated by the high incidence of gangrene of the heels of those boys who rode out of the Reservoir area on jeeps or weapon carriers

**Adrenal Gland and Clotting** —The adrenal replacement or stimulating substances such as cortisone or ACTH appeared to cause increased thrombotic tendencies Cosgriff et *al* [17] reported 7 per cent thrombosis

**Antibiotics and Their Effect on Clotting** —The widespread and routine use of antibiotics is commonplace One effect of these drugs is the sterilization of the intestinal tract. The flora disappears and the normal organisms are absent or present in minimal amounts Vitamin K is synthesized as the result of the bacterial action of the organisms normally present in the bowel This vitamin is a neutralizer of the coumarin compounds and is important in clotting This cessation of its production increases the bleeding tendency Since the success of modern bowel surgery is dependent

upon the reduction or removal of intestinal organisms prior to opening the intestinal tract, the increased possibility of hemorrhage must be kept in mind In like manner, the anticoagulant drugs, if utilized, will have greater effect, and may require only one-quarter to one-half the dose to maintain a therapeutic level Unfortunately, these findings are variable and inconsistent Without question, they are responsible for some of the so-called "anticoagulant drug accidents."

**Clotting and Malnutrition—Carcinoma, etc.**—Carcinoma and diseases of the organs which contribute to the digestion, such as the liver or pancreas, as well as conditions causing vomiting or diarrhea, all produce an undernourished physical status In our studies of anticoagulants, we found that such patients are more sensitive to the anticoagulant drugs A smaller dose will create a higher prothrombin level It follows, therefore, that such patients are more susceptible to bleeding and have a defective coagulating mechanism The change arises after only a few days of malnutrition It also accompanies the poor nutrition which follows the overuse of alcohol. The effect of the absence of vitamin B appears contributory, but this has not been demonstrated conclusively.

## THROMBITIS (THROMBOPHLEBITIS, PHLEBITIS)

Since there are many gaps in our knowledge of clotting which remain to be filled by studies in physical and biochemistry, a true classification cannot be made From the clinical standpoint, however, we can differentiate two types of clotting—the inflammatory and the non-inflammatory The former is designated as *thrombitis* and the latter a *thrombosis* We thus eliminate such terms as thrombophlebitis, phlebothrombosis, periphlebitis, and bland, quiet and silent thrombosis, all of which are confusing to clinical minds

The causes of clotting of either type probably are similar fundamentally Thrombitis or the inflammatory type is more often associated with infection or trauma It occurs most often in those patients who have had previous venous pathology The two types of clotting will be discussed separately as to symptoms, diagnosis, and treatment

**Symptoms.**—The symptoms of *thrombitis* are those of inflammation These are *redness*, *heat*, *swelling*, *pain*, and *tenderness* which follow the course of the vein involved The severity of the symptoms depends upon the degree of inflammation At times, it extends along the full course of the vein Inflammation often extends into the surrounding tissues There is clotting and induration palpably present in the vessel wall The *leukocyte count* is elevated and an increased *sedimentation rate* is present The *pulse* and *temperature* are elevated

The condition may be mild or extensive There is a secondary *edema*, and if the condition persists sufficiently long, a compensatory secondary lymphedema develops in which there is pitting edema with pigskin-like changes If the brawny induration continues, a lymphatic block occurs and an elephantiasis develops The veins distal to the inflammation become dilated The patient may be extremely ill Continued activity aggravates the process Later symptoms are edema and dilated veins, which are collateral vessels and may be mistaken for simple varicose veins

Approximately a year after such an attack ulceration occurs in from 20 to 30 per cent. This ulcer is usually above or near the malleoli. It is characterized by extreme pain and resistance to all types of therapy. There is an associated fungus infection in nearly every instance. Secondary infection of the ulcer follows. Skin changes include pigment deposits, redness and desquamation.

**Pathology.**—The inflammation in thrombitis arises in or near the vein and extends in the wall of the vein with the clot propagating along the intima. There is an invasion of leukocytes, edema and other signs of inflammation. There is a precipitation of protoplasmic elements forming a grey white thrombus (white thrombus) and when this is complete the blood thus trapped forms a clot-cast of the vessel (red thrombus).[40, 87, 91] The part the platelets play is important as is their tendency to cohesiveness. This is still not fully understood.[44–47, 86] The wall of the vessel becomes extremely thickened and after a time there may be efforts to recanalize the clot. The clot which at first is soft is soon invaded by cells and phagocytes and then replaced by fibrosis.

There may be considerable periphlebetic changes early in the form of inflammation, with edema and the invasion of cells secondary to an inflammation. Eventually the endothelial wall is destroyed and replaced by fibrosis. The lesion may be localized at one or many points or at times may be slowly propagated along the walls. Occasionally the inflammation develops in different veins in the body, the so-called migrating thrombitis.

The relationship between migrating thrombophlebitis and malignancy has been noted by many observers.[84, 88] In questionable cases recurring phlebitis may be an indication for surgical exploration and often points the way to a diagnosis of malignancy. In some patients a multiple and recurring phlebitis migrates from part to part and organ to organ. It has been reported in the brain[78a, 80] and at times is uncontrollable and fatal. If the lesion is resistant to anticoagulant therapy one should look further for an underlying cause.

**Treatment.**—The treatment of thrombitis is divided into the relief of the symptoms of the thrombosis and of its complications.

A. Primary Treatment of Thrombitis.—*Vascular Packs.*—Patients with thrombitis are much more comfortable and resolve better when external warmth is applied. The heat should be in the form of a warm pack after protection is applied to the skin. The leg is wrapped in warm wet towels saturated with saline solution and 2 to 4 hot water bags are inserted. The whole leg is then covered, including the foot, with rubber sheeting. Burns must be prevented.

The water is tested each time with a thermometer to see that the temperature is no higher than 92° F. In this respect hot water bags are safer than electric pads because the hot water bags invariably cool and if not applied too hot cannot burn. There is danger of an electric shock with the electric pad and wet towels.

These packs can be left on from four to six hours. The pain and spasm are relieved by this pack. The pack should be applied so that the patient can move freely.

*Reflex heat* also reduces spasm. It can be provided by immersion of the arms in warm water or the application of heat to the abdomen.

Warm Sitz baths are effective. The attendant fungus infection can be eliminated by foot soaks of potassium permanganate (1:10,000 to 1:30,000 strength).

*Movement* of the part is encouraged, first in bed and later when the patient is up. Full weight bearing on the legs should be delayed until the inflammatory process has subsided. This must not interfere with active and passive motion of the leg, as this motion prevents the stasis and trauma to the vessels which result from being in one position too long.

*Sedatives.*—Due to the pain, morphine or Demerol may be required in many instances, later being replaced by codeine and aspirin. Whiskey also helps relieve the pain. Milder sedatives can be used in less severe attacks.

*Anticoagulant Therapy.*—Anticoagulant therapy is indicated in every patient with thrombitis of severity. It should be instituted unless there is a definite contraindication to its use. Among the factors limiting its employment must be included inadequate laboratory facilities necessary for its control. The great danger of thrombitis is its extension. Anticoagulant therapy correctly applied will prevent further clot involvement in most patients. Details of the management of anticoagulant therapy are given on pages 654 to 658. The treatment should be continued from fourteen to twenty-one days in the average patient. Other patients require longer therapy, and in certain instances ambulatory anticoagulants have been prescribed for an indefinite time. (See pages 659 to 660.) Repeated recurrences and thrombitis migraines are examples of such indications.

*Interruption of the Sympathetics.*—Sympathetic interruption in the treatment of thrombitis was first advocated by Leriche forty years ago.[45a]

The technic for sympathetic nerve block has been described in the chapter on Interruption of the Sympathetics, page 494. Any well-trained physician can perform such a block. The first, second, third, and sometimes the fourth lumbar ganglia on the affected side should be injected with an anesthetic solution. The solution usually used is 5 cc. of 2 per cent procaine hydrochloride in each ganglion. At times, a longer anesthetic is used to prolong the sympathetic effect. A continuing block is effected by inserting a polythene tube as the needle is removed. (See page 498.)

The effect of the sympathetic nerve block is to break the sympathetic reflex synapse by anesthetizing the ganglia. This synapse neurogenically is established by afferent stimuli to the ganglia from the affected vessel, the ganglia in return sending efferent stimuli to other points, both in the affected vessels and in the collateral vessels, thus causing the spasm. (See Figure 146, page 454.) The chemical theory of reflex spasm has already been discussed on pages 256 and 487. It has been proven that this spasm is the cause for both the edema and pain. When this synapse once is broken, the reflex action often does not re-establish itself. This has been proven clinically by showing skin temperature rises in these limbs for as long as three days and occasionally for a week after a block, although the procaine anesthetic lasts only an hour.

One to six blocks may be required to obtain the desired effect. (Four in our series.)[63] Approximately 75 per cent obtain relief of their symptoms.

In some recurrent and continued thrombitis surgical interruption of the sympathetics has been effective Chemical interruption of the ganglia may help if blocks cannot be performed (See pages 490 to 494.)

*Combined Sympathetic Blocks and Anticoagulants* —For ten years the author has used sympathetic blocks in addition to anticoagulant therapy in the treatment of inflammatory clotting Nearly 600 patients have been so treated [a 67] In no single instance has there been a complication of a hemorrhagic nature While the addition of the anticoagulants makes hemorrhage a possibility, it is the feeling that post-sympathetic block bleeding is a technical error and not a complication because of anticoagulant therapy If one is rough repeatedly jabs needles around the vena cava or lumbar veins or particularly if sufficient experience has not been gained on cadaver blocks hemorrhage will occur with or without the anticoagulant factor The beneficial results of the combination of these treatments when properly applied outweigh the dangers In experienced hands the co-application of these treatments is advocated They are complementary to each other

In the inflammatory clotting patient two pathologic patterns are present. One is the tendency for the clot to propagate and spread to other parts of that and other veins and the spasm factor which is initiated and maintained by the inflamed clot This mechanism has been discussed in the chapters on Occlusive Arterial and Spastic Diseases and will not be repeated needlessly This reflex is one of Nature s defense mechanisms to prevent injured vessels from bleeding and to squeeze out clots to maintain an open vessel to carry blood again later This mechanism is helpful when the vessel is injured openly but when the injuring agent is an intraluminal clot the mechanism becomes pathologic and continuous

The therapeutic problem is to prevent the clot spread and to eliminate the spasm These two effects can be achieved by a combination of anticoagulant therapy and the sympathetic blocks That the two methods can be used at the same time and to the advantage of the patient without jeopardy to the patient s well being by bleeding is shown by our experiences with the combined therapy of this lesion In a series of 560 patients who were on antithrombotic therapy over 2100 sympathetic nerve blocks were performed without a single post block hemorrhage [a 67] During this same interval four massive retroperitoneal hemorrhages were seen these hemorrhages occurring after blocks were performed on patients who were not receiving any antithrombotic drugs Obviously care must be exercised in performing sympathetic blocks but our series indicates that if this precaution is taken the mere fact that the patient has a lowered prothrombinemia does not mean he will hemorrhage from the block [a]

When local or blood cultures are positive antibiotic therapy always is administered Penicillin Sulfonamides Aureomycin Terramycin Chloromycetin Streptomycin and other antibiotics are given as indicated

The side effects of antibiotic therapy must be remembered Such drugs at times cause anaphylactic and allergic reactions These drugs also sterilize the bowel of certain organisms and serious gastrointestinal reactions follow their prolonged use Since vitamin K is synthesized by the action of the bacteria of the bowel its normal production may be inter

rupted Renal complications may ensue. Such therapy should be discontinued after five days except in extraordinary circumstances

*Ligation* —Ligation and resection of the vein in the presence of *inflammation* as a rule is contraindicated Two fatalities occurred from multiple and repeated embolisms after this procedure, one in fifteen minutes and one in twenty-four hours after such a ligation was performed

The inflammation extends much further in these vessels, at least microscopically, than one suspects from the line of redness Operation on such an inflamed vessel adds trauma to the inflammation Clotting may occur above the point of ligation or division and, from this uncontrolled site, embolism may result

There are certain selected patients in which ligation is indicated despite inflammation Minor vein inflammations which recur and do not respond after a considerable period of conservative therapy are an example Another indication for ligation is when suppuration results in uncontrolled embolism In patients with repeated emboli, ligation often is effective Repeated attacks of pneumonitis or pneumonia often are the result of emboli In such cases, ligation of the vein should be performed despite the fact that inflammation is present

When embolism does occur in thrombitis the embolus is usually a small one This is due to the fact that the inflammation keeps most of the clot adherent to the intima wall and prevents extension and propagation

B SECONDARY TREATMENT OF THROMBITIS AND ITS COMPLICATIONS — (1) *Edema* —Edema will be present for a time after thrombitis, and it will continue, depending upon the degree and extent of the inflammation, the type of active treatment received, and the aftercare

To treat the edema, it is important to realize that *elevation* of the part above the heart level will remove the fluid *Adequate support* should be applied and maintained The maintenance of this adequate support cannot be overemphasized Too many patients with thrombitis are permitted to discontinue their support after a short time If this is allowed, the edema will persist

The drainage from the skin has depended upon these superficial veins which are now blocked due to the thrombitis If complete recovery is to be expected, drainage to adequate or deeper veins will be necessary This is supplied by (*a*) elevation of the part above the heart level each time fluid accumulates, (*b*) support to the extremity whenever it is dependent and (*c*) the muscular action of walking In most instances, it is advisable to continue support with an Ace type of bandage or an elastic stocking for at least a year The mechanics of this therapy must be understood and explained thoroughly to the patient The idea is to keep the subcutaneous spaces free of fluid for a long time If fluid accumulates, tissue spaces for it are formed and these will persist If these spaces are kept emptied of fluid and the support is applied, the tissue spaces do not form and the area may scar closed

(2) *Ulceration* —Ulceration is a frequent complication in patients with thrombitis It occurs particularly after thrombitis of the superficial femoral vein This ulceration occurs most often at the time when recanalization of the superficial femoral vein is occurring [48] These ulcers are fre-

quently multiple, sometimes small, extremely painful, shallow and always surrounded by edema and an angry appearing dermatitis. This is often of fungus origin containing streptococci and other secondary invaders.

The treatment of thrombitic ulcers is both local and general. (See also the Treatment of Ulcers on page 781.)

*Local Treatment.*—The part should be kept *elevated* and in most instances *exposed to the air* at room temperature. Infection should be eliminated by *soaks* with saline and potassium permanganate solution 1:10,000 to 1:30,000 to destroy the invading fungus.

As streptococci can be cultured from most ulcers, both local and general antibiotic therapy should be applied. Cultures and sensitivity tests will determine the type of antibiotic most suitable.

Elevation, soaks and exposure will give these ulcers a clean base. Ointments, dyes and antiseptics are not advocated as they cause tissue maceration. Supporting bandages such as elastic or rubber boots are of some value to achieve healing but at times give the patient a false sense of security. Their use may be contraindicated for this reason. Good results with vitamin E have been reported, but similar results may follow any therapy. Temporary interruption of the sympathetics with such drugs as tetraethylammonium chloride may help healing.

*Adenylic Acid Therapy.*—Following the original work of muscle injection of adenylic acid in Hodgkin's disease, it was found that the pruritis of that disease was relieved. Thereafter, a series of patients with ulcers and dermatitis of venous origin were treated in the Vascular Clinic of St. Vincent's Hospital of New York City. The itching and weeping would stop and the status of the skin improve under such therapy.[72] These ulcers would close. If the injections were discontinued, the lesions recurred. It was recognized that such therapy was an adjunct to the treatment of ulcer complications. It is now used to prepare the patient for definitive surgery. The substance is prepared as a gel and has a sustained action. It is given in 1 cc. intramuscular injections every other day.* It seems certain that in these ulcers the individual cells are lacking in adenylic acid. Replacement therapy, therefore, *is an aid in healing.*

*Operative Treatment.*—When the ulcer has become bacteriologically clean and there is absence of any inflammation in the vein, operative treatment is required. This treatment is delayed until six to nine months after the inflammation has subsided. The operative therapy may require only resection of the causative veins in the superficial or deep venous systems. In other instances, the ulcer must be excised and skin grafted. In a few the sympathetic system must be denervated to maintain healing. (See page 507.)

## SUPPURATIVE THROMBITIS

This condition, fortunately, is rare. It results from a thrombitis which continues to suppuration or the organization of a clot originally not infected but which develops sepsis secondarily. It may occur anywhere in the body but most often is found in the pelvic veins. The occurrence of

* Prepared as "My B-Den," made by Ernst Bischoff Co., Inc.

infection of the pelvic veins after childbirth, miscarriage, or an abortion which becomes infected makes the female pelvis a frequent site. In some patients the clots become secondarily infected from septicemic sources.

Suppurative thrombitis may be divided into that affecting the peripheral veins and the condition occurring in the pelvis, since the latter is a distinct entity.

1. **Suppurative Thrombitis in Peripheral Veins.**—This lesion is a rare continuation of the inflammation of the leg veins. It occurs most often in the saphenous or in the superficial femoral system. In the latter the diagnosis may be masked for a time and osteomyelitis may be suspected. Usually such a lesion soon involves the superficial veins and the signs of redness, streaking and marked edema are added to the fever, chills, leukocytosis and malaise. Localized abscesses occur. Septicemic emboli and septemia are complications. The prognosis usually is grave.

TREATMENT.—*General.*—Antibiotic therapy is indicated, and where cultures are obtained, sensitivity to the antibiotic drug should be made. The correct antibiotic is selected. Small, fresh, whole blood transfusions increase the patient's resistance. The therapy, in general, should be directed against the sepsis. Anticoagulants may help in preventing clot propagation. The treatment should be vigorous and continued.

*Local.*—Locally the part should be kept in warm wet packs and weight bearing prevented. Motion of the part should be encouraged on a passive basis.

*Ligation.*—If there is evidence of progression or embolization, interruption of the vein between the primary site and the lung should be performed. The decision as to this procedure must be made before the condition has spread. Ligation may help in localization, although the prognosis must be guarded. The site of ligation usually is the inferior vena cava. Sympathectomy should accompany the ligation. (See page 639 for Vena Cava Ligation and Sympathectomy.)

2. **Suppurative Pelvic Thrombitis.**—This condition begins following some pelvic intervention where there has been infection. An obstetrical delivery, pelvic abscess, an abortion, dilation and curettage or application of radium are common initiators of the condition. Secondary to the pelvic sepsis, the veins develop purulent clots. These show early, and continued embolic tendencies and death is usually due to this continued septicemia or secondary abscesses, particularly in the lung. The clots soften with bacterial digestion and are thus ready for movement with further necrosis. The emboli usually are small and multiple in contradistinction to those of thrombosis. Thus death results from sepsis rather than the mechanical arrest of blood flow as occurs from a blockage of the pulmonary artery.

DIAGNOSIS.—The diagnosis is made on the history of pelvic intervention, the chills, fever and increased pulse rate, chest x-rays (positive in 16 per cent),[15,16] abdominal and pelvic examination with signs of pain, tenderness and peritonitis, and, at times, the findings of thrombosed vaginal or pelvic veins (60 per cent).[16]

TREATMENT.—A. *Medical Treatment.*—Large and continued courses of antibiotics should be given with a combination of blood transfusions and anticoagulant therapy. Abdominal distention should be kept minimal by

enemas and an indwelling duodenal tube with suction Attention should be paid to complications in the renal or pulmonary systems In many patients this regimen combined with a no touch program for the pelvis will result in subsidence of the process In approximately 1 out of 4 such therapy is unsuccessful In the other patients the infection progresses to suppuration the clots break free and embolisms result Death is due to the septicemia or the multiple embolic abscesses

B *Surgical Treatment* —This is indicated for any patient who does not respond to conservative measures A transperitoneal incision is made The inferior vena cava is isolated and ligated in continuity below the renal veins The left ovarian vein which drains into the left renal vein is ligated near its entrance to that vessel [52] In one group of 70 patients reported by Collins[14-16] 90 per cent recovered This operation has been performed in 8 instances by our Clinic without mortality

## INTRACRANIAL VENOUS THROMBOSIS IN THE PUERPERIUM

This condition relatively is rare but occurs often enough to be a separate entity There are 6 reports of the condition in the literature [39, 42, 50, 51, 73, 77]

*Etiology* —The exact cause of an intracranial venous thrombosis in the puerperium is not known One theory is that it is a retrograde venous embolism from the pelvic veins [50] In such event the clot would be carried from the pelvis through the valveless vertebral veins to the superior sagittal sinus This clot then would propagate The possibility that the tight abdominal binder usually worn after delivery closes off the vena cava and facilitates entrance into the vertebral vein is one theory The strain of a bowel movement may be an etiologic factor Another thought is that the thrombosis in the cerebrum is primary In this theory the damage occurs during labor as the result of an increase in the plasma fibrinogen the thrombocytes and more adhesiveness of the platelets when large numbers of new cells are released from the bone marrow The intracranial venous pressure is raised during labor and this with blood stasis due to the intra-abdominal tension and forced expiration may cause the local brain lesion [42]

*Symptoms* —The lesion develops from the fourth to the twenty-first day after a normal delivery There is headache local or general convulsions and mono- or hemiplegia There may or may not be loss of speech If the superior sagittal sinus is obstructed there will be papilledema. The symptoms vary naturally with the degree of involvement.

*Diagnosis* —The diagnosis is made on the symptoms If the existence of the syndrome is known the diagnosis is not difficult. In the past, the condition has been diagnosed erroneously as encephalitis or 'late eclampsia. The lesion has also been called cerebral arterial thrombosis Others have stated that the condition was due to a cerebral embolus from the pelvic or leg veins For this diagnosis to be valid one would have to assume that there was a patent foramen ovale

*Prognosis* —The mortality varies from 30 to 56 per cent.[42,73] If the patient survives the initial severe symptoms recovery may be rapid and complete

*Treatment* —In general, the treatment is an expectant one which includes good nursing care. The anticonvulsive drugs control such signs Paralysis requires care of the limbs If the cerebral spinal pressure is high, small amounts of it should be removed daily. Hypertonic glucose may be helpful and whole blood transfusions repeated every two and three days may help the patient's reactions [39,50 51 75]

The anticoagulant drugs have their advocates and opponents The problem is complicated by the recently enlarged uterus and delivery These drugs can be used if careful and accurate laboratory measures are available and maintained The danger of cerebral hemorrhage with overdose of anticoagulant therapy must be kept in mind

Antibiotic Therapy —Antibiotic therapy must be extensive and continuous Where possible, a culture should be obtained A pelvic culture may show the susceptibility of the organism to the drugs and the type of antibiotic to employ

## VENOUS THROMBOSIS (THROMBOEMBOLISM)

**Etiology.**—This has been discussed under Mechanism of Venous Clotting on pages 608 to 616

**Symptoms.**—The symptoms of thrombosis, unfortunately, are not too apparent Often the lesion is not diagnosed Even if the symptoms are not too apparent, a diagnosis can be made in 80 per cent of the patients if thrombosis is considered

Many of the signs described for thrombosis are very late ones and a fatality may occur before some of the signs are obvious

The symptoms which occurred in our series[70,71] of 90 cases of massive pulmonary embolism are presented in the order of their frequency of occurrence

1 *Pain* is present in all cases of thrombosis. This is a difficult symptom to evaluate because each patient differs in his threshold for pain The pain will be in the popliteal space or in the calf At times, the pain may begin at the tendo achillis and gradually extend up the leg Movement of the part usually increases the pain

*Homan's sign*, described as pain in the calf of the leg on dorsal flexion and extension of the foot, is a valuable but late sign Any condition causing pain on motion of the calf, such as a boil or a pulled muscle, will produce a positive Homan's sign A mild continuing pain from the knee to the ankle, more severe on movement of the foot, should make one suspicious of thrombosis

2 *Tenderness* is present usually in the popliteal space and sometimes in the calf With the extension of the process, the pain and tenderness will follow the course of the vein up the thigh This path of extension should be examined

3. *Edema* is present, but it is *mild* The clot, as it propagates along the intima, at first, does not occlude the vessel completely When it does extend and occludes the vessel, the pressure behind the clot builds up suddenly and embolism will occur at that time If the embolism does not occur and the clot remains lodged, then there will be massive edema. Such

edema will be of the pitting type and marked like the so-called post partum milk leg

4 *Cyanosis or duskiness* is an early sign and is valuable when the part is compared directly with its fellow The cyanosis is distal to the clot and may not be extensive Dependency of the two parts will greatly increase the cyanosis on the involved side If both legs are involved the color of the feet can be compared to the hands



Fig 195 —Extensive inflammatory venous thrombosis Onset left leg extension to right leg, and to superficial abdominal vein Multiple pulmonary emboli Ligation of inferior vena cava. Subsequent extension to mammary veins and emboli through superior vena cava Eventual recovery

5 *Sentinel Dilated Veins* (Pratt)[illegible] There are three small branch veins just below the knee on the medial side of the leg which open into the lesser saphenous vein and then communicate with the popliteal vein These are dilated early and consistently in thrombosis in the popliteal area At times this is the earliest and only diagnostic feature These veins are believed to dilate first because they are the closest vessels to the popliteal vein which is so often blocked and therefore they are involved early

6 The patient's *temperature* usually is elevated and this sign is inconsistent with the patient's general condition The temperature of the leg generally is raised If reflex spasm occurs the part may be cold

7 The *pulse rate* is increased In a report of a group of patients with pulmonary embolism only 1 out of 5 had a pulse rate below 90 and 3 out of 4 had a pulse rate considered abnormal for their surgical status at that time.[illegible] Anxiety may increase the pulse rate

8 The *respiration* increases with the temperature and pulse but rises suddenly if there is an embolism

9 The *blood pressure*, in general, is low In the fatal embolisms just mentioned, the systolic pressure was below 140, despite the fact that the majority of the patients were old This factor, in itself, may be important in pathogenesis from the stasis standpoint

10 *Pulmonary Embolism* —A small embolus, in the absence of other sources, is a diagnostic sign of thrombosis A small embolism will occur in 1 out of 3 patients prior to a major embolism If there is not adequate treatment when embolism does occur, there is a 50 per cent chance that another will occur and an incidence of 1 in 3 that the subsequent one will be fatal



FIG 196 —*Dilated "sentinel" veins, three small veins which enlarge over the anterior tibia early in patients with femoral thrombosis These veins dilate early because they drain into the popliteal vein which is one of the earliest sites of clotting*

It is certain that many undiagnosed embolisms occur It is our belief that 9 out of 10 pulmonary complications following general surgical operations are due to embolism and not due to local pleuritis, pleurisy, bronchitis, or pneumonia, as they are usually diagnosed. Emboli occur most often in the lower lobes of the lungs If they lodge near a bronchus, hemoptysis occurs early If the embolus is near the periphery, pain will be an early sign There may be tenderness on percussion Central emboli may be difficult to diagnose

11 *Abnormal Fear* —Many of these patients sense that *something* serious is happening to them This has occurred so often that when an

intelligent patient suspects or fears a clot it is sufficiently indicative for us to consider therapy Such awareness should stimulate a search for a clot

**Pathogenesis of Thrombosis** —This subject has been discussed under Thrombitis and will not be repeated needlessly [14, 44, 55] See pages 608 to 616

*Summary* —The classical theory of blood coagulation has been modified Pathologic clotting can be summarized as follows Whether these changes are initiated chemically or physically alone or in combination is not established Many components make up blood Normally *in vivo* these elements remain separate In clotting however most of them develop a cohesiveness It is evident that there is a variability in the tendency to clot from time to time from place to place in the body and that clotting depends on whether the body is in a diseased or normal status It is apparent that prothrombin liberates thrombin very slowly The addition however of the accelerator globulin causes prothrombin to more quickly convert prothrombin to thrombin Many other accelerator factors have been described [72] such as component A (Quick) [71] Factor Five and Six (Owren[64]) serum prothrombin conversion accelerators[1, 18, 60] and calcium These may be similar factors In addition thromboplastin is necessary Its mode of action is unknown some believing an activator controls it while others suggest that it has an inhibitor activity [33, 72] The final reaction is between fibrinogen from the blood s protein and thrombin to form fibrin The electrical forces of the substance may play a part This has been defined as zeta potential Zeta potential is calculated on the streaming potential and is actually the potential difference between the wall potential charge and that of the interior of a solution arbitrarily placed at zero The part that platelets play has been known for a long time The increased stickiness of such cells has been brought to our attention recently [17] Platelets increase in number and adhesiveness as the result of trauma operation and an obstetrical delivery Normally red and white cells do not agglutinate nor do they adhere to the inner surface of vessels The blood cells move in the center the plasma surrounding it The platelets being smaller are forced to the surface If the vessel wall has a break in continuity the platelets adhere first If the stream slows a sludge-like material develops This sludge then becomes adherent to the endothelium more cells join it, and eventually it occludes the lumen the white thrombus This white head then is a nucleus and this is propagated by an accumulation of red cells and other blood components the so-called red thrombus

**Treatment.**—The management of venous thrombosis involves both prophylactic measures and active treatment These methods may be medical and surgical or both

1 **Prophylactic Measures** —Prophylactic procedures are of utmost importance in preventing the occurrence of thrombosis

(a) *Early Ambulation* —Stasis tissue injury and clotting factor changes follow *lying in bed* *Bed rest* therefore is as great a cause for pulmonary embolism as an operation This is true particularly, in the traumatic group of patients who have fractures or who are immobilized in casts or extension apparatus for a long time

The patient should be kept *ambulatory* up to the time of operation and should be allowed to become ambulatory as soon thereafter as it is possible. We performed over 6,000 operations on 5,100 patients under local anesthesia with immediate ambulation. There was no patient who developed a thrombosis.[70,71]

The importance of keeping the patient mobile cannot be overemphasized in the prevention of thrombosis. Should this be impossible due to some general condition, active motion of the legs in bed should be continued, if the patient is able. If this is not possible, then regular passive motion should be ordered. The program we follow is to tell surgical patients to move their legs 1,000 times after operation. The importance of this program even in medical patients in the prevention of clot propagation is shown by the recent ambulatory (armchair) treatment of coronary patients.[47]

(*b*) *Tobacco*.—The use of tobacco should be eliminated in all individuals who are to undergo an operation or who are bedridden for any length of time. This requires emphasis. The use of tobacco by all over forty years of age probably is contraindicated.

(*c*) *Fungus Infection*.—Fungus infection should be eliminated as far as is possible. A mild fungicide should be ordered and continued regularly to prevent this possible source of thrombosis by secondary infection. Such caustic drugs as salicylic and benzoic acid in this prevention should not be used.

(*d*) *Overweight*.—Obesity should be reduced prior to any operative procedure. The delay of an elective operation for one or two months for weight reduction will be reflected not only by a reduction in thrombosis but in all postoperative complications. It is likely that the increased lipemia in obesity is a stimulation to venous clotting.

(*e*) *Anemia*.—Anemia should be corrected. In this status the cellular elements of the blood are not in their normal proportions and that may be the cause of the clotting. Anemia also stimulates the hematopoietic system with the outpouring of juvenile blood cells similar to that seen in hemorrhage. Intravascular clotting occurs more often under these circumstances.

(*f*) *Hemorrhage*.—For the reasons stated under Anemia, hemorrhage should be prevented as far as it is possible.

(*g*) *Cardiovascular Status*.—The cardiovascular status should be corrected if possible. Efforts to control both the anemia and the cardiovascular status of the patient will be reflected in the morbidity and the mortality. One-third of our fatal pulmonary embolisms occurred in patients with cardiovascular disease.

(*h*) *Minimal Operative Trauma*.—The pictures of sludged blood (see page 612) impress one with the necessity for minimal operative trauma. Rough retraction, blunt dissection, long exposure of tissues, and slow operating must be reflected in more sludged blood and, consequently, in more thrombosis.

(*i*) *Restriction of Intravenous Therapy*.—With modern anesthesiology has come the increased use of the veins for pentothal, glucose, etc. When

it is necessary venipuncture should be performed with fine sharp needles If the solution is to be run for considerable time the needle should be replaced by a fine plastic tube Irritating chemical solutions should not be injected and the solutions should be as isotonic as possible The arm veins should always be selected in preference to a leg vein which is much more prone to become inflamed The anesthesiologist should be reminded that supplemental intravenous injections should be used only when necessary Minor procedures or those performed under spinal anesthesia do not have to be supplemented by pentothal At times it appears that the pentothal is given for the convenience of the anesthesiologist rather than for the patient s comfort In addition the pentothal makes the patient sleep postoperatively and thus delays the early ambulation or motion

(j) *Restrictive Bandages or Positions* —Straps tourniquets cuffs bandages and adhesive tape should be applied with the thought of their possible constriction effect on the veins The surgeon should examine such restraints himself Pain the warning sign of constriction is eliminated under anesthesia

(k) *Varicose Veins* —Patients with varicose veins have venous stasis This should be controlled by bandages and active motion At times the elimination of the veins by ligation prior to a major procedure is worthy of consideration Such veins should not be used for venous therapy Pressure on such legs should be avoided

(l) *Temperature* —There is less thrombosis in patients who are in a normal and unvarying environment than where it is cold or very warm

(m) *General Measures* —Deep breathing exercises after operation delivery or where bed rest is prolonged should be routine Constriction of the abdominal wall by girdles or binders should be prevented Blood replacement and hydration to keep the proportion of cellular contents to the liquid elements in the blood normal will result in a lowered incidence of thrombosis Any infection should be actively treated Other prophylactic measures such as the administration of thyroid extract the high carbohydrate-low protein diet etc have not been effective and have certain disadvantages [84]

(n) *Antithrombin* —The use of an antithrombin has been advocated It has not been proven that alphatocopherol with or without calcium prevents thrombosis

(o) *Anticoagulants* —These substances have a definite place in the prophylaxis of thrombosis The use in the patient with a fresh operative wound must be tempered with judgment. If hemorrhage is suspected the anticoagulants are contraindicated This applies to wounds where hemostasis is incomplete or where drainage is instituted It is our practice to use these drugs prophylactically only where we suspect thrombosis may occur and when there is no contraindication In most patients the anti coagulants can be added to the regimen with safety one day after operation or when the drain has been removed (See Antithrombotic Therapy pages 651 to 666 )

A NONSURGICAL THERAPY —2 **Active Treatment.**—Once the diagnosis of thrombosis is made, the type and length of treatment depend upon the stage of the thrombus pathogenesis and its site or sites (See pathogenesis page 608) If there is massive edema, the danger of a fatal embolus from the affected leg probably is over An intolerance to anticoagulants, tendency to hemorrhage and familial embolic history may be factors in the decision

Our feelings as to the treatment of thrombosis have changed greatly since 1948 [71] The indications for surgical interruption of the veins have become fewer and more definite With increasing experience and better anticoagulant drugs together with earlier diagnosis we can reduce the number who require operative intervention There is a definite indication, however, for resection of the vein proximal to a clot Such indications can best be summarized by posing the following question

"In this given patient is embolism likely to occur due to the underlying disease, previous history, site of clot and the length of time it has been present?"

If the clot is a fresh one and is at a site which indicates it is large enough to close off a pulmonary artery or a large section of the lung, we believe it is best to place a mechanical barrier, *i e*, ligation between such a clot and the lung

*Anticoagulant Therapy*—If the condition is diagnosed when the clot is small, early and likely in the calf veins, anticoagulant therapy will be instituted The treatment will be directed towards preventing clot propagation and relieving symptoms caused by the clot already present No operation will be performed in these patients at this time unless the clot propagates, embolism occurs or there is some contraindication to the institution or maintenance of the anticoagulant regimen In the majority of such patients the treatment will resolve the process in eighteen to twenty-four days This therapy with warm vascular packs, elevation and mobility of the part and support when the patient is on the leg, often is all that is necessary There is no conclusive evidence that the clot already present will be dissolved by the anticoagulants but the extension will be controlled If the thrombosis is diagnosed, or the patient seen late, for example, two weeks after the onset of the condition, the same regimen should be followed Contraindications to this therapy mean a vein ligation These are. 1. If the patient has had an embolus from a leg vein and survived it, we believe that a resection of the common femoral vein as a mechanical block to further embolism may be indicated Embolisms have occurred in such patients even after adequate anticoagulant therapy If the thrombus is present at the operative site, a thrombectomy is performed at the same time

2 When anticoagulant therapy has not stopped the process or has not prevented an embolism, an operation is indic[illegible] A continuation of the process is shown by continued pain, prop[illegible] the local clot, progression to the other [illegible] an embolism

3 In those pa[illegible] have had re[illegible] [illegible]eks of thrombosis and/or embolism, [illegible] an indication [illegible] of the vein

4 In those patients where hemorrhage may occur or is likely from the se of anticoagulants ligation of the involved vein is indicated Examples f such conditions are peptic ulcer insecure operative hemostasis and leeding from pulmonary tuberculosis

5 Where advanced renal or liver damage contraindicates anticoagulant herapy ligation of the involved vein should be done If there is liver or idney damage it is illogical to give oral antithrombotic drugs Severe ver disease interferes with the production of prothrombin, and in such ases the ingestion of vitamin K causes no response The patients with idney disease also develop liver damage and have an increased tendency o bleed They eliminate the drugs irregularly

*Vitamin Deficiency* —Vitamin K deficiency produces a hypoprothrom inemia. It appears that vitamin K is synthesized in the intestine by the ormal bacteria therein Sterilization of such bowel by antibiotics as for xample in the preparation for major surgery may reduce the vitamin K itamin C deficiency appears to influence the hypoprothrombinemia in uced by anticoagulants

6 If the clot is high and there is an intolerance for or a poor reaction o the anticoagulants ligation of the inferior vena cava should be done

7 *Blood Dyscrasia* —Such conditions as aplastic anemia purpura and eukemia have an increased bleeding tendency, probably due to release of eparin Polycythemia although having a tendency to occlusion also roduces increased bleeding

8 *Late Pregnancy* —There are two dangers in the use of anticoagu ants. Hemorrhage may occur at the placental site In addition the drug may accumulate in the mother and may cause hemorrhage after delivery The second danger is that some of the drugs pass through the placenta to he baby This may cause a hemorrhage in the child prior to or immedi- tely after the birth

9 *Subacute Endocarditis* —These patients have an inherent tendency o bleed and the use of anticoagulants is contraindicated The previous ombination of anticoagulant and antibiotic therapy in this disease has een discontinued in most clinics

B SURGICAL THERAPY —1 **Common Femoral Vein Resection.**—When here is an indication for surgical interruption of the deep veins of one leg, ommon femoral vein resection is our operation of choice This vessel is hosen rather than the superficial femoral vein as used in some clinics be- ause we wish to block also the femoral profunda vein which drains many f the muscles of the calf These calf veins are the source of many of the riginal clots which become large thrombi and embolisms McLachlin re- orted on the ineffectiveness of superficial femoral vein ligation [64]

Resecting the common femoral vein also blocks the saphenous vein

There should be only a slightly greater incidence of residual edema when he common femoral vein rather than the superficial femoral vein is ligated With adequate support after the operation the common femoral veins can e ligated nearly as safely as far as residual edema is concerned as can the uperficial femoral vein The massive edemas that have occurred after such ein ligations are in those patients in whom an inflammatory clot was pres-

ent in the superficial veins This inflammatory clot blocked the collateral vessels Such patients, in most instances, did not need their femoral veins ligated

*Anesthesia* —A local anesthesia is effectual and safe A general anesthetic is contraindicated because of the possibility of aspiration embolism due to the straining of deep breathing during the induction or second stage of anestheia Spinal anesthesia, while an ideal one, requires considerable manipulation of the patient to place him in the correct position Embolisms

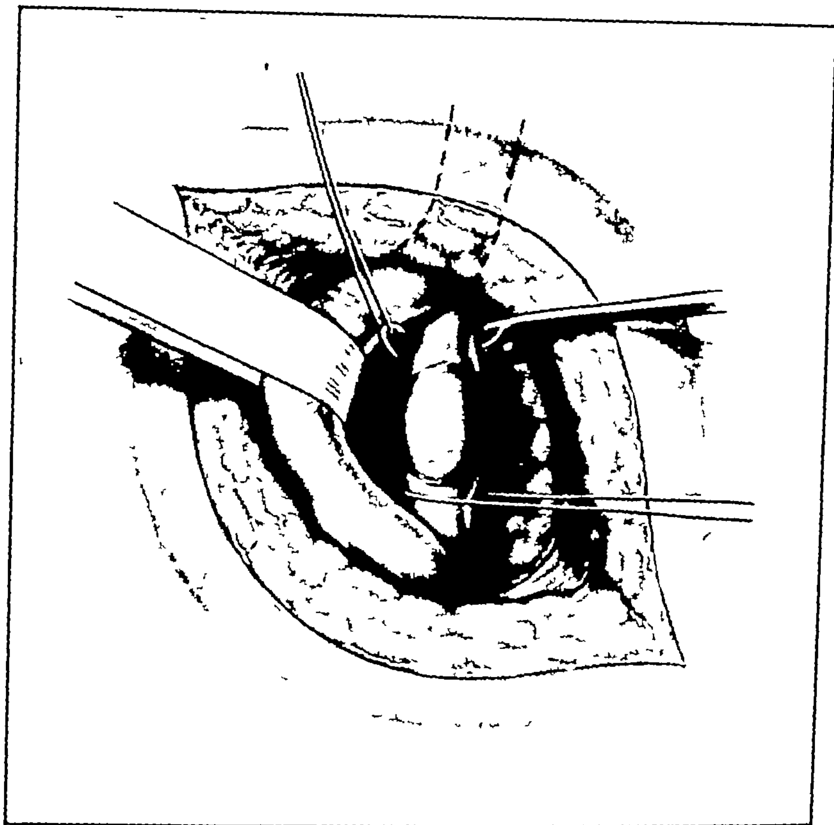


FIG 197 —Anatomy of the common femoral vein and artery This shows that in the groin area the common femoral artery lies directly over the femoral vein This is significant because sometimes the artery in spasm may be mistaken for the vein This figure shows the femoral artery retracted and the femoral vein thus exposed

have occurred when the knees were "jackknifed" preparatory to a spinal anesthesia While spinal anesthesia can be given in the prone position with the patient on the abdomen, this, again, requires manipulation

*Operative Technic.*—A vertical incision 3 to 4 inches long is made through the deep leg fascia just medial to the junction of the great saphenous and femoral veins The femoral artery is located very superficially and lies directly over the femoral vein in this area and both the vein and the artery are contained in a combined sheath The sheath is dissected free with as little trauma as possible The common femoral vein is a very short vein and, approximately 2 inches above the point at which the saphenous vein joins, it dips into the pelvis to become the iliac vein Dissection with a

blunt hemostat away from rather than into the vein and artery will separate them from the surrounding tissues Where there has been some inflammatory process present innumerable adhesions may be found

The dissection of the femoral artery from the femoral vein sometimes is *difficult* The artery may go into spasm and resemble the vein This artery has been ligated instead of the vein for this reason and this technical point must be kept in mind at all times

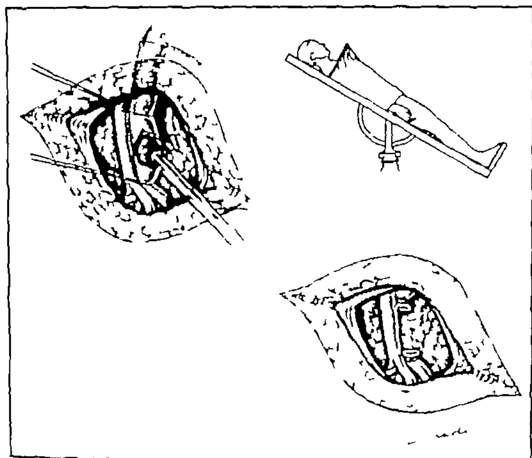


Fig. 108.—Thrombectomy and ligation of the common femoral vein The first picture shows the artery retracted and the femoral vein brought into view with guy sutures. A longitudinal incision has been made and the clot is being removed with suction The second picture shows the vein ligated and tran fixed Insert shows the position of the patient on the table Fowler s type of position reduces the possibility of embolism during the thrombectomy (*Pratt courtesy New York State J Med*)

A small McBurney type of retractor makes an excellent ligature carrier The usual ligature carrier or aneurysm needle is a traumatizing type of instrument sometimes with a sharp point It may penetrate the vein rather than go *around it* *The McBurney type of retractor is dull and will* lift a part of the vein (See Figure 107 ) The dissection can be completed *with the use* of a dull hemostat.

When the vein has been dissected free and is cradled in the loop of the McBurney retractor a No 00 silk suture can be placed through the loop of the retractor and pulled around the vein The vein is then mobilized for a distance of three-quarters of an inch

With extra help to control the guy sutures, a longitudinal incision is made in the common femoral vein. As soon as the incision is made, the edges are grasped with mosquito-type hemostats and everted The guy sutures prevent serious bleeding If a thrombus is present, it is teased out with a smooth forceps or with the suction catheter After the thrombus



FIG 199 —Clots removed from the common femoral vein of a patient who was having multiple pulmonary embolisms

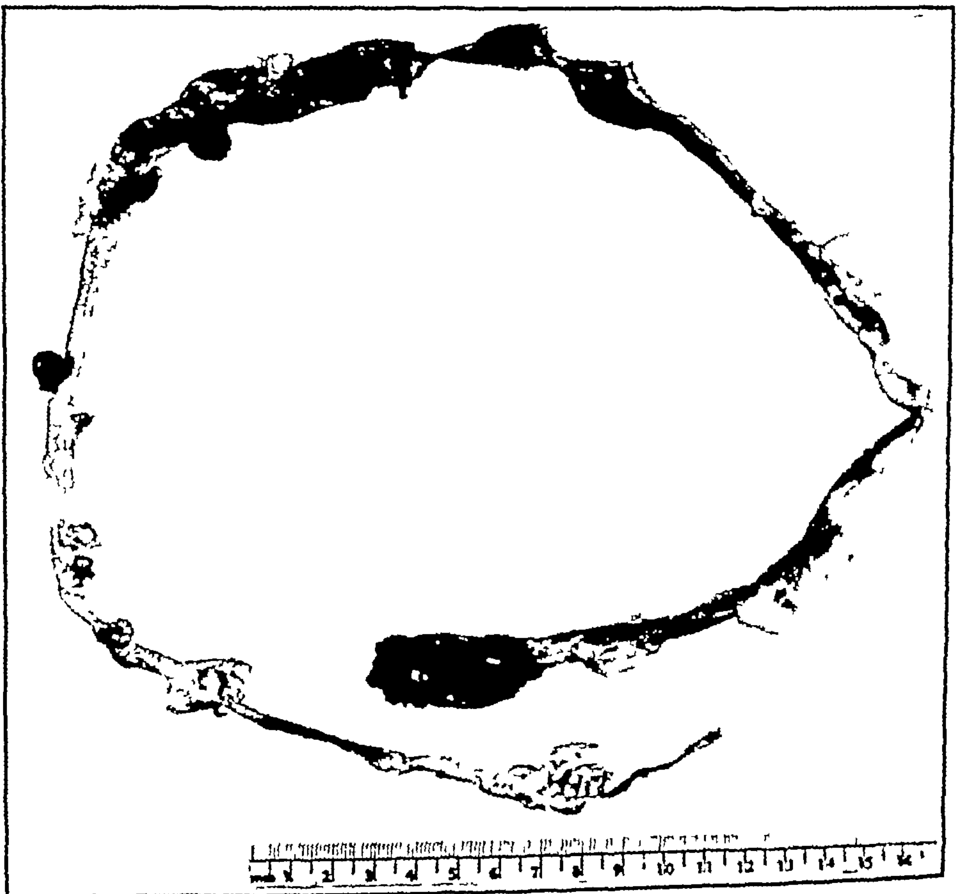


FIG. 200 —Thrombosed vein excised *in toto* Many months of conservative therapy had not resolved the process Superficial femoral vein ligated at the same time

has been removed, the catheter or glass drinking tube is inserted through the rent and passed up into the iliac vein or the vena cava This process is repeated until such time as a free and continuous flow of blood from above is obtained. Spasm may reduce this flow

When one is certain that there is no thrombus above, any distal clot is removed, if possible, in the same manner Both the proximal and distal

ends are transfixed and ligated. When the vein is sectioned, the proximal end will retract into the pelvis. Care must be exerted to prevent the retraction from occurring before the ligatures are well tied. There may be considerable venous pressure on these vessels, and without a transfixion suture there would be danger of hemorrhage. The wound is then closed by covering all of the silk sutures with a subcutaneous catgut suture to close the fascia, and we use fine wire for the skin.

*Postoperative Care.*—Care of the patient after a common femoral vein resection consists of the administration of antithrombotic drugs as discussed on pages 651 to 666, the active movement of the part, control of any associated fungus infection, elevation of the foot of the bed, walking at once, and support to the part when the patient is on it. Warm vascular packs are helpful.

*Complications of the Common Femoral Vein Resection Operation.*—(1) *Reflex Arteriospasm.*—Rarely reflex arteriospasm is a complication. This has been reported from time to time in the medical literature, but has occurred after femoral vein ligation in only two instances. Both of these

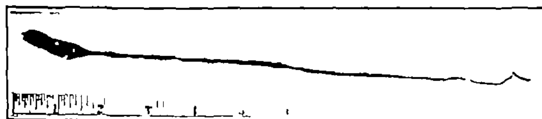


Fig. 201.—Thrombus removed from saphenous femoral junction. Thrombus lying free in vein.

patients had a marked arteriosclerosis. Trauma alone rather than any reflex action may have caused the arteriospasm.

Should arteriospasm occur, all of the measures to overcome spasm should be employed at once, including the lumbar sympathetic nerve blocks.

If serious arterial occlusion occurs after common femoral vein resection, it is possible that the femoral artery was included in the ligation. This is not impossible because this artery goes into spasm, is cord like, superficially placed, and it can be readily ligated instead of the vein by the inexperienced surgeon.

(2) *Femoral Vein Resection and Gangrene.*—Up to 1950 we found only 5 authors reporting gangrene of the extremity following venous obstruction or inflammation in the American literature.[4,12,16,18,21,22] The French literature contained many references, beginning with that of Haldanus in 1593.[22] Cruveilhier, 1862, mentioned it, but as late as Buerger's writing[11] it was stated that extensive obstruction only could precipitate this lesion. Fontaine and deSouza Pereira[23] produced the condition experimentally. DeBakey and Ochsner[18] found 24 cases in a review published in 1949, and Haimovici added 27 patients in 1950.[21] It is apparent that marked thrombosis of nearly all of the venous channels is necessary for this lesion to develop.

An underlying arterial disease probably must be present When gangrene develops following a vein ligation, technical errors must be considered as the most likely cause In the absence of such mistakes it is likely that there was a massive venous clotting of the superficial veins to which femoral vein ligation was added Embolisms remain a possible cause While the possibility of such complications exist, they will be extremely rare in the hands of qualified surgeons

(3) *Edema.*—Edema will occur in from 4 to 10 per cent of the patients so operated The degree varies with the extent of clotting of collateral circulation and the postoperative treatment This complication is not a contraindication to the operation, as vein ligation is performed to prevent death The edema can be kept minimal by the following

a Support.—This support must be adequate and maintained from the toes to the knees for sufficient length of time This support is applied as soon as the patient arises and is best cared for with Ace type of bandages The bandage should include all of the foot, heel and ankle besides the leg, and should be evenly but not tightly wrapped If the bandage becomes tight it should be loosened and the leg elevated, and the bandage later re-applied This is an important point and must be stressed, as some swelling occurs for a time in all patients

b Elevation —The legs should be elevated by raising the mattress 8 to 10 inches each night This effectively drains the edema since the part is above heart level In addition, elevation is necessary during the day whenever the leg swells This elevation must be continued until trial shows no swelling occurs without it

c Sympathetic Interruption —Some patients develop causalgic syndromes with swelling, coldness, heaviness of the part, paresthesia and pain Many of these will respond to sympathetic blocks In others sympathectomy is necessary and usually is curative (See Postthrombotic Syndrome, page 674 )

d Warm Vascular Packs —These will help in reducing the swelling and some of the symptoms of pain and heaviness They should be used from four to five hours each day (See page 617 )

e Abstinence from Smoking —This has been discussed before and is a dictum

f Active Motion.—Walking with support aids in reducing the edema by muscular action This should be graded within the limits of the patient's circulation and never to the fatigue stage Walking or swimming in warm water is a valuable aid

(4) *Ulcer* —This is a definite complication of venous thrombosis and is discussed under Postthrombotic Ulcer, page 667 (See chapter on Postthrombotic Syndrome, page 667 )

(5) *Postthrombotic Syndrome* —This complication results in 1 out of every 5 patients (See chapter on Postthrombotic Syndrome, page 667 )

2 **Resection of the Iliac Vein.**—This operation has been replaced by the inferior vena cava ligation because its exposure and ligation is technically easier In addition, if one iliac vein is involved, often there has been some extension retrogradely to the other iliac vein There are a few instances, however, in which the iliac vein must be ligated

3 **Ligation of the Inferior Vena Cava.**—*Indications* —The indications have been discussed under Femoral Vein Ligation They are recent thrombosis bilaterally thrombosis in the iliac vein embolism from a single or double site and embolism after superficial or femoral vein ligation The operative technic for ligation of the inferior vena cava is essentially the same as the exposure for lumbar sympathectomy on the right hand side

*Anesthesia* —Spinal anesthesia is the anesthesia of choice and its employment facilitates the exposure A hypobaric type permits immediate table break (See page 32 )

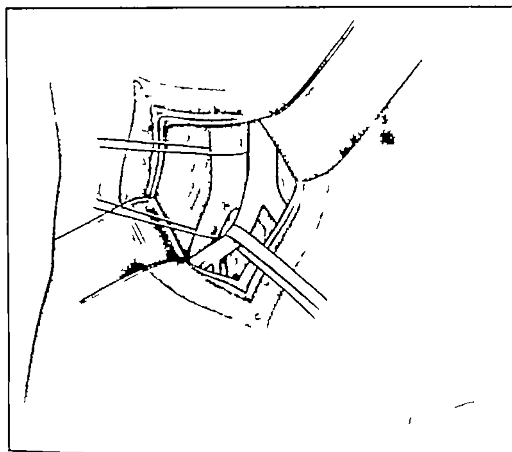


FIG. 202.—Operation for the exposure of the iliac vein or vena cava. Retroperitoneal approach. Transverse incision preferred (Pratt courtesy of New York State Jour Med.)

*Technic* —The position of the patient is extremely important and if correct makes the operation more simple (See Fig 156 ) The patient should be placed with the right side elevated approximately 40 degrees so that the viscera falls to the left lower side away from the operator Breaking the table at the operative site to hyperextend this area gives adequate room between the crest of the ilium and the lower rib edges The patient s right arm is swung up and to the opposite side grasping the opposite side of the operating table and thus pulls the lower ribs out of the operative field The leg on the right side is kept straight but the left leg is doubled under the right leg in the Simm s fashion with a pillow between the two limbs. A strip of 3-inch adhesive centering over the greater trochanter and

the bent knee anchors the patient A transverse skin incision is made 2 inches above the crest of the ilium, with a muscle-splitting division of the external oblique, internal oblique, and transversalis muscles The transversalis fascia is divided near its lateral edge rather than its medial edge where the peritoneum is adherent The peritoneum and its contents are then pushed forward to the midline with two mounted wet sponge forceps. This maneuver is aided, if necessary, by tilting the table to the left side and also by raising the lumbar rest

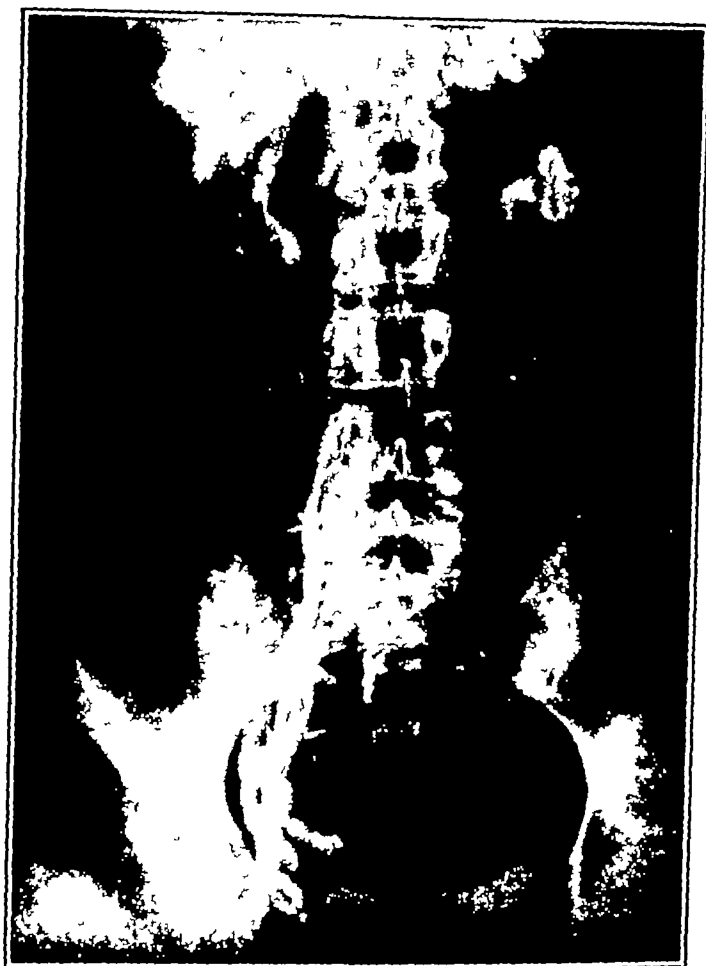


FIG 203 —Venogram two years after inferior vena cava ligation for multiple pulmonary emboli Note original pathology in iliac and femoral vein with valve incompetency Partial recanalization of inferior vena cava

Once the psoas muscle has been exposed and the peritoneum has been separated from it, a Harrington retractor will hold the viscera forward Small Deaver retractors in the superior and inferior part of the wound displace the rest of the viscera and the extraperitoneal fat Exposure in this instance is important and depends upon the assistant opposite the operator, who holds the Harrington retractor displacing the peritoneum and its visceral contents

After the viscera have been displaced, the vena cava is dissected free from its surrounding fat covering with a mounted peanut sponge stick and a dull hemostat The surgeon must ascertain that he is below the renal

veins Once there is a line of cleavage around the vena cava laterally it is separated from the aorta medially The curved small McBurney retractor is of great value in placing the silk ligature around the vena cava This instrument can be held much like a fountain pen and with the retractor on the opposite side of the gloved finger the dull edge of the McBurney retractor can be worked around the vena cava When the other end of the instrument appears a No 0 or 00 silk suture can be placed in it and the vena cava can thus be mobilized The silk suture held on tension will permit a second or third silk suture to be passed

Unless there is evidence of a thrombus at this operative site the vena cava may be *ligated in continuity* inasmuch as a mechanical block is all that is necessary The opening of the vena cava for thrombectomy is an operation fraught with danger because of its inaccessibility and because of the tremendous collateral blood supply which exists in this vessel When thrombectomy is required adequate help good retraction and a good light are imperative The vessel then may be opened and the thrombus removed after which ligation is performed as mentioned above The use of brain clips to control any annoying bleeding from small vessels or branches of the vena cava is of great help Transfixion sutures are required

The wound is closed by permitting the viscera to fall into place A No 1 chromic catgut is used on the transversalis muscle and interrupted No 32 steel wire on the internal oblique and the external oblique muscles which are closed in layers without drainage

*Postoperative Care* —Care of the patient following ligation of the inferior vena cava is essentially the same as it is after femoral or iliac vein resection (See page 635) Rarely is there marked swelling because of the adequate collateral circulation The antithrombotic drugs are used in all cases of major vessel ligation The operation is not to replace the use of antithrombotic therapy but is used in addition to it

*Sympathectomy with Vena Cava Ligation* —Whenever the inferior vena cava is ligated lumbar sympathectomy should be performed This operation is advisable because the possibility of reflex vasospasm is markedly reduced it is relatively simple and the exposure is the same In addition the incidence of a postthrombotic syndrome is much less when sympathectomy is performed The procedure consists of dissecting free the second and third ganglia and removing the first and fourth ganglia by exeresis as detailed on page 505 in the chapter on Sympathectomy

The vena cava has been ligated indiscriminately too often in the past years. There are indications but these are limited[illegible] The fact that the operation has been restricted to selected cases by most of the large vascular clinics is evidence of the operation s potential danger

*Complications after Vena Cava Ligation* —Edema has been reported by many observers

It is not necessarily a complication unless there has been superficial venous clotting and inflammation We have not had massive edema in any of our over 40 inferior vena cava ligations Some edema must be expected and accepted The other complications such as ulcers (one-third) and other trophic changes as well as arterial spasm vary from 10 to 30 per cent and depend to a great extent upon the previous venous pathology and the support therapy[illegible]

## THROMBOSIS OF THE UPPER EXTREMITIES—SUBCLAVIAN AND AXILLARY VENOUS THROMBOSIS (EFFORT THROMBOSIS)

The peculiarities and the variations from the normal in the anatomy at the point of junction of the neck and axillary regions have been described (see page 472) The exposed position of the subclavian vein, especially in its middle and third portions, makes it subject to undue stress and strain on violent exercise or movements of the neck, thorax, or shoulder girdle

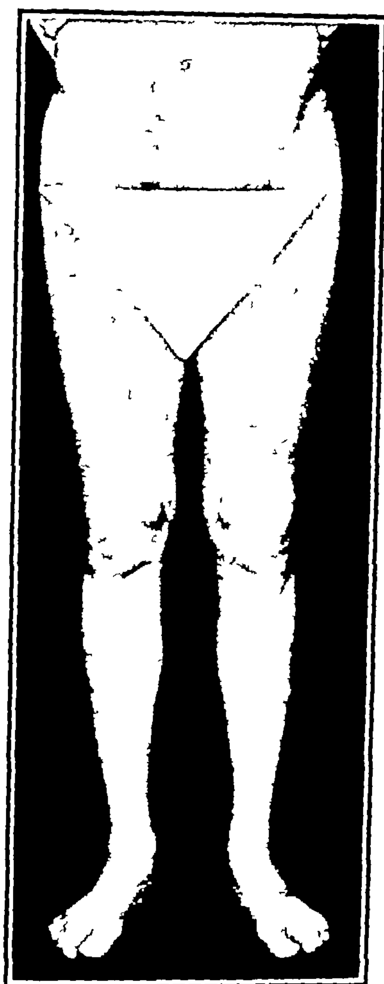


Fig 204 —Postoperative picture inferior vena cava ligation for multiple pulmonary emboli Note absence of swelling Sympathectomy performed at the same time

**Anatomy of Venous Circulation in Upper Extremity.**—*The Veins of the Upper Extremity* —The veins of the upper extremity are divided into the superficial and deep, the two systems anastomosing frequently

(*a*) *Superficial Veins* —The digital veins are composed of the dorsal and volar sets The dorsal vessels form three metacarpal veins which end in a plexus on the back of the hand The vein from the radial side is joined by the dorsal veins of the thumb and this anastomosis becomes the cephalic vein The ulnar part has a fifth digital connection and this combination

becomes the basilic vein The volar digital veins drain into a plexus on either side of the wrist The cephalic vein an extension of the radial part of the dorsal veins ascends the arm and ends in the axillary vein below the clavicle An accessory cephalic vein joins it below the elbow The basilic vein which is the opposite of the cephalic ascends laterally and joins the brachial to form the axillary vein The volar surface is drained by the median antebrachial vein It ascends the front of the forearm to join the basilic vein

(b) *Deep Veins* —The deep veins form the comitantes of the arteries They are situated on either side of the arteries They make up the volar arches by a junction of the digital veins They correspond to the branches of the arterial arches The volar digital veins open into the superficial arch and the volar metacarpal into the deep volar arch In the forearm the deep veins are the comitantes of the radial and ulnar arteries and are continuations of the deep and superficial arches At the elbow they form the brachial veins These latter are on either side of the brachial artery They join the axillary vein at the subscapularis muscle or occasionally the basilic vein The axillary vein is made up of the basilic and brachial veins It receives many branches and the cephalic vein joins it near the teres major muscle The axillary vein begins at the lower border of the teres major muscle and extends to the first rib where it becomes the subclavian vein The axillary vein is a continuation of the basilic vein The axillary vein receives the brachial veins near the lower border of the subscapularis muscle and the cephalic vein near where it becomes the subclavian vein

As the subclavian vein extends through the neck from the first rib it is in relation with the clavicle and subclavius muscle in front but is separated from the subclavian artery behind by the scalenus anticus muscle The vein is joined by the external jugular often the anterior jugular and at its junction with the internal jugular, the subclavian vein on the left receives the thoracic duct and on the right the lymphatic duct At the sternal end of the clavicle the subclavian vein joins with the internal jugular vein to form the innominate vein The two innominate veins are short vessels without valves They meet just below the cartilage of the first rib at the right border of the sternum to form the superior vena cava

Any abnormality such as a large rib a hypertrophied muscle particularly the scalenus muscle a fibrous band or any calcified node affects the normal relationship of the axillary or subclavian veins and subjects them to increased trauma or torsion In addition an unusual position or a continued pressure due to some type of work may cause pressure and clotting

The scalenus anticus muscle plays an important part This muscle arising from slips of all six cervical vertebrae descends as a flat ribbon-like muscle to insert near the scalene tubercle in the first rib Normally the subclavian vein is superficial to it Any unusual motion of maneuver that causes undue strain of the scalenus anticus muscle may so compress the subclavian vein (against the clavicle) as to cause a thrombosis Subclavian or axillary vein thrombosis may follow A hypertrophied scalenus anticus muscle or an abnormal first or cervical rib may be such an underlying cause

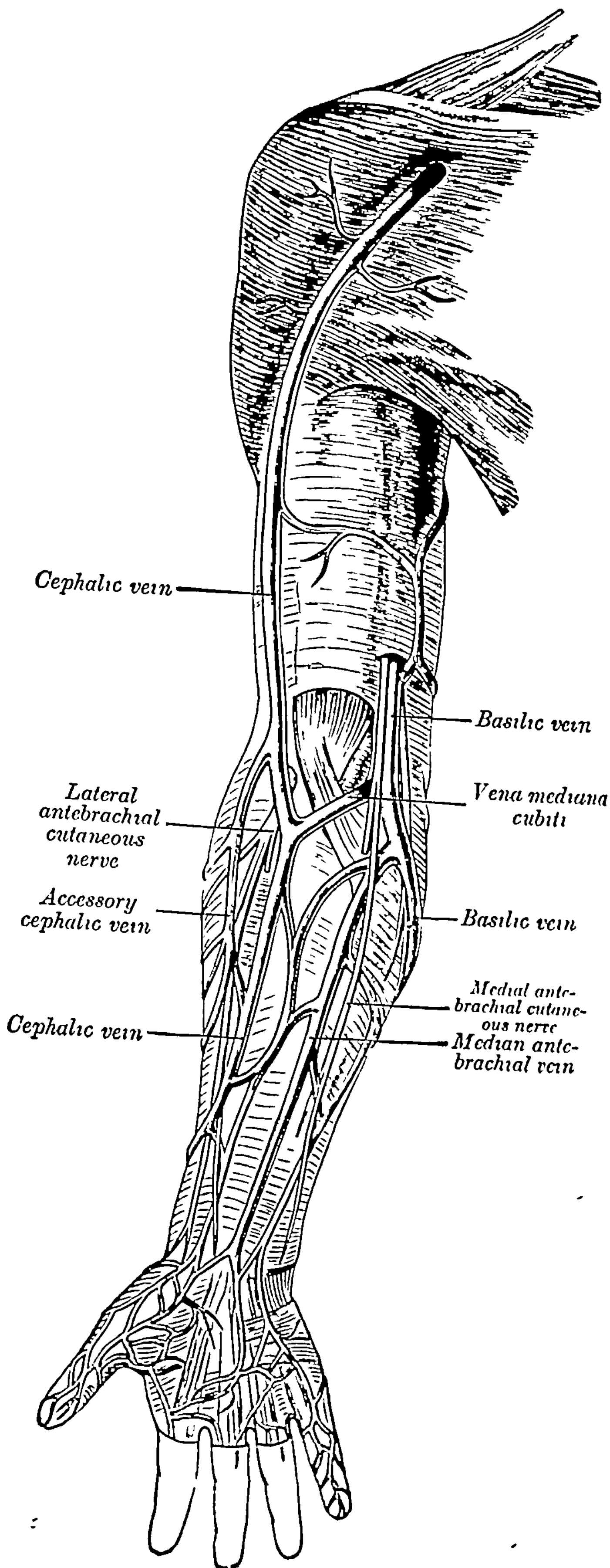



FIG 205 —Anatomy of venous system, upper extremity (Gray's Anatomy)

**Etiology** —The anatomical etiology has been discussed Subclavian or axillary vein thrombosis most often is seen in patients who overdevelop their scalenus anticus muscles at puberty time The lesion is more prone to occur also with a change in occupation to one which causes extensive neck muscle development Persons who suddenly begin to perform work that requires them to extend their arms above their shoulders are more subject to such thromboses Professional weight lifters, wrestlers painters

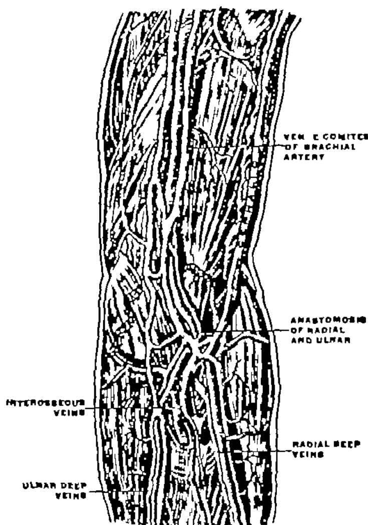



FIG 206 —The deep veins of the upper extremity (Bourgery)

plasterers ceiling cleaners and others who have large hypertrophied neck muscles also may be subject to this condition Unusual positions such as painting ceilings cleaning windows working under a car pitching hay or threshing grain may precipitate thrombosis in the susceptible individual Such a thrombosis also may be secondary to malignant invasion *In this* respect carcinoma of the breast is the usual cause A massive thrombosis or edema of the arm after a radical mastectomy should be considered of malignant origin until proven otherwise.

**Exciting Cause.**—There is usually some trauma which precipitates the condition. This may be not too severe, but it is sufficient to cause a sudden and sharp exertion on the scalenus anticus muscles. It is also likely that a spasm of the subclavian vein arises due to compression by the subclavian muscle tendon alone or in conjunction with the scalenus anticus muscle.[52] In others there may be direct trauma to the axillary or subclavian vein. Sufficient injury to the endothelial walls of these vessels to cause stasis and trauma is necessary to produce the thrombosis. In a few, the thrombosis may be of unknown origin. Occasionally the thrombosis may de-

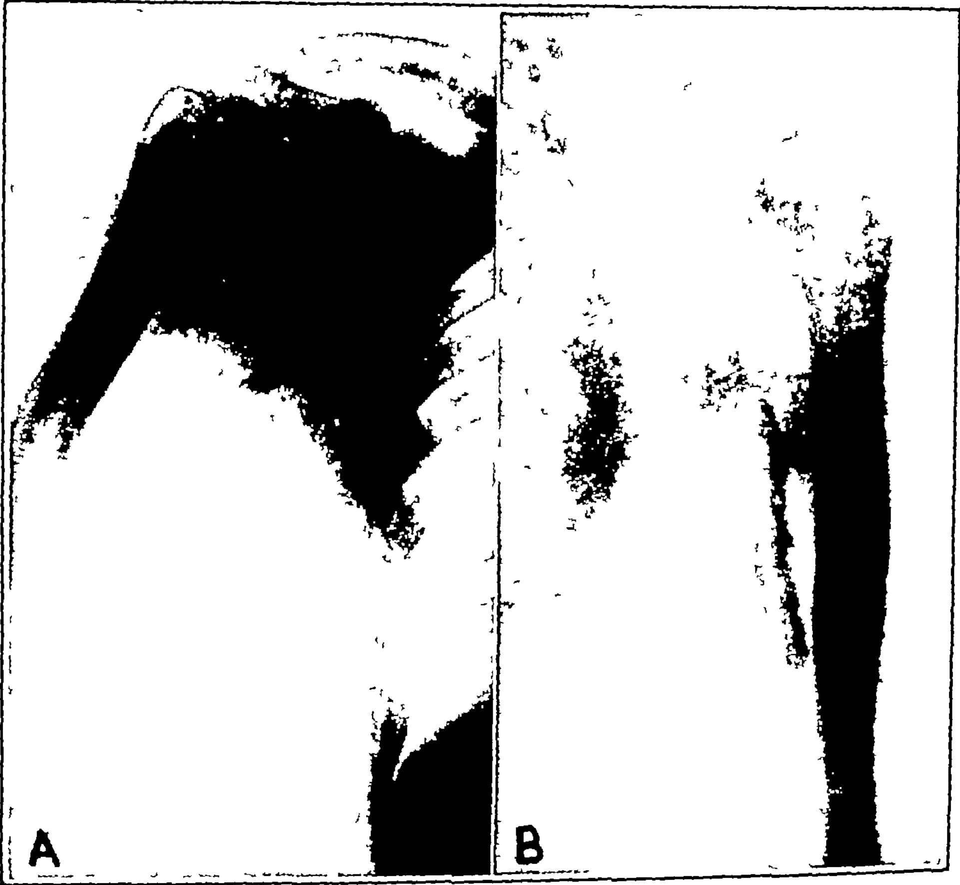



Fig. 207.—*A*, Venogram of a normal axillary and subclavian vein. Collateral vessels do not fill. *B*, Subclavian and axillary vein thrombosis. Back pooling of blood into collateral vessels.

velop following a thrombitis in other parts of the body, the so-called phlebitis migraines. Fracture, hypertrophy or developmental anomaly encroaching on the space between the first rib and the clavicle may cause the thrombosis.

**Symptoms.**—The symptoms of axillary or subclavian vein thrombosis are usually sudden, but in a few the symptoms develop over a period of days. There is usually *pain* at the site of the clotting. This is followed by *swelling* and *duskiness* of the part. The *cyanosis* of the arm may be extreme. The *veins* distal to the thrombus become *dilated*, and collateral vessels appear. There may be such large dilatations as to simulate a caput

Medusa In some only the large veins over the shoulders and chest wall dilate

As a rule the *edema* is massive This may be of the pitting type The swelling may be reduced by elevating the part The affected arm becomes warmer as the blood is suffused Elevation will improve the color of the extremity, and dependency increases the cyanosis There is a heaviness and fullness and marked restriction of motion Pulmonary embolism may occur but this is rare compared to thrombosis of the lower extremity

Following malignant infiltration the edema does *not* subside

**Pathology**—The clot usually develops at the site of the trauma or may develop exactly where the scalenus anticus muscle has compressed the subclavian vein This clot propagates distally to some extent and may involve some collateral vessels (See Pathogenesis of Venous Clotting on page 608)

**Prognosis.**—In the absence of malignancy the prognosis is good Recanalization usually occurs This usually takes from three to six months It can be aided by elevation of the part warm packs and the use of sympathetic nerve blocks to relax the spasm in the collateral vessels Graded exercises and mild physiotherapy measures may help to stimulate this collateral venous circulation

Recurrence is to be expected until the causative pathology has been relieved It is not unusual for the patient to give a history of repeated thromboses once one has occurred The prognosis as to life is good inasmuch as the incidence of embolism is exceedingly low

**Treatment.**—The immediate treatment is that of elevation of the part abstinence from overuse and warm packs Sympathetic nerve blocks of the stellate ganglia will help materially in relaxing the spasm The use of antithrombotic drugs to decrease the tendency to propagation of the clot is indicated This has been described in the chapter on Antithrombotic Therapy on page 651 Contrast injections to determine when recanalization has occurred will be helpful prognostically but should be delayed until the clot is thoroughly adherent.

**Scalenotomy**—Because of the tendency for thrombosis to recur anterior scalenotomy should be performed The technic of scalenotomy has been described under Scalenus Anticus Syndrome (see page 476) The scalenus anticus muscle should be divided near its insertion on the first rib This procedure can be performed under local or endotracheal anesthesia Trauma to the subclavian vein must be avoided The operation should be delayed until recanalization has occurred in the main vein and until all possibility of the clot movement has been eliminated While scalenotomy does not relieve the swelling or large collateral veins present on the shoulder or arm it does remove the cause for possible repeated thromboses. In some instances the obstruction to the vein occurs under the clavicle This is best determined by venogram If such obstruction exists, excision of the entire subclavian muscle is indicated This can be done at the same time as a scalenotomy by continuing the incision over the clavicle and laterally just beneath this bone[42] The costoclavicular ligament is divided to facilitate the dissection

**Other Surgical Treatment.**—(1) *Thrombectomy*—Thrombectomy rarely is necessary in conditions of this kind. It should be performed only if the condition progresses or embolism occurs

(2) *Resection of the Collateral Veins*—After the main vein has recanalized, these enlarged collateral veins may be pathologic In some, it is necessary to resect these collateral veins In others, they may be locally sclerosed This should not be done until there is evidence that the deep vein has recanalized and is functioning satisfactorily This can be proven with intravenous angiography

(3) *Sympathectomy*—In some, there is a resultant causalgia with residual swelling. This is not unlike the postthrombotic syndrome seen in the lower extremities These patients complain of coldness of the part, mild or marked edema, duskiness, and repeated attacks of swelling.

If sympathetic nerve blocks give relief from the swelling and pain and if the pain is of the causalgic type with anesthesia and parasthesia, a thoracic sympathectomy should be considered The operation should be reserved for those who do not respond to other types of therapy It has been necessary to perform sympathectomy to alleviate this condition 7 times in the last 32 scalenotomies we performed for subclavian and axillary vein thrombosis. The occasional unpredictable failure of sympathectomy must be kept in mind

(4) *Resection of Bone*—Where fracture, hypertrophy or developmental anomaly has caused the bone to encroach on the space normally occupied by the subclavian vein, resection of such bone may be required This may be accomplished by ronguering the faulty bone from either the clavicle, rib or the vertebræ At other times, removal of a section of the clavicle or first rib or the entire bone may be required [48b]

(5) *Malignancy*—Where the cause of the thrombosis is a malignant infiltration around or into the veins or lymphatics, surgical intervention rarely helps *X*-ray or radium may be palliative Surgery usually is contraindicated

## OBSTRUCTION OF THE SUPERIOR VENA CAVA

Obstruction of the superior vena cava occurs rarely. It may develop as a complete or incomplete block The incomplete blocks cause few symptoms and many are found at autopsy Hussey reported 35 cases in 1946 [38] Most of the compressions of the superior vena cava are caused by cancer in the chest or aortic aneurysm [38 63] These obstructions progress and are usually fatal The incomplete occlusions prognostically depend upon the cause and the site of obstruction (above or below the azygos vein) and the development of collateral circulation Glushien's[27] patient lived thirty-six years Mediastinitis of syphilitic or other origin is a frequent cause

**Symptoms.**—The symptoms are due to restriction of drainage by the superior vena cava The signs are edema and cyanosis of the head and neck, the *syndrome of dizziness and syncope during exercise*, prominence of the eyes and edema of the conjunctiva and the phlebographic evidence of the block Dilated veins are a part of the picture

**Treatment** —Most of these conditions cannot be remedied surgically Early antiluetic therapy will help the syphilitic A removable tumor is correctible Some of the aneurysms producing this syndrome respond to operation [7] Babcock s operation has been helpful in some The mediastinal decompression operation of Graham rarely is applicable [29 38]

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Chapter

# 35

# ANTITHROMBOTIC THERAPY (ANTICOAGULANT THERAPY)

## *Anti Clotting Substances—Treatment of Hemorrhage Due to Antithrombotic Drugs*

Very few therapeutic additions to medicine and surgery actually are new. Anticoagulant therapy joins the antibiotics, cardiac surgery, the revolutionary changes in anesthesia and blood and fluid replacement on a quantitative and qualitative basis as an elemental addition. Surgeons have been slow in accepting this measure and the new field that it opens. Internists are far ahead both in the control and application of the antithrombotic principle. This is understandable, as the surgeon must keep in mind a fresh operative wound. Sufficient experience has accumulated, however, to make this attitude no longer tenable. Certain fundamental controls are necessary, and when these are applied, these drugs are not only safe but at times a lifesaving addition to the surgical armamentarium. These drugs are as safe as their user.

The term "anticoagulant drugs" utilized for the past ten years has become suggestive of hemorrhage to many surgeons. We purposely term these drugs *antithrombotic* to denote their therapeutic use rather than to describe the occasional accident which follows their misapplication.

These antithrombotic drugs prevent propagation of clots and possibly may dissolve some clots. Correctly utilized, this therapy prevents propagation of small clots—embolism—and therefore death. These drugs also will eliminate the necessity of surgical intervention in many patients. There can be no inflexible or definitive type of therapy. The treatment depends upon the stage and extent of the pathology and the response to therapy. The surgeon who decries all antithrombotic therapy probably is unskilled in its use and to cover such failure cries "Wolf!" Equally dangerous is the internist who knows that there is a soft clot present in the femoral or iliac vein but refuses to suggest that surgical ligation block that clot from extension to the heart or lung. Both of these extremists will have deaths which are preventable. In the early days of the use of these drugs the fresh surgical wound was placed first on the list of contraindications to antithrombotic drugs. This forced the surgeon to use operative measures. The failure of such a one-sided program has been reported. Over 1600 prophylactic ligations on postoperative patients in one hospital in a year resulted in many vascular cripples. Separating the medical from the surgical patients and treating only the former by anti

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Chapter

35

# ANTITHROMBOTIC THERAPY (ANTICOAGULANT THERAPY)

## *Anti-Clotting Substances—Treatment of Hemorrhage Due to Antithrombotic Drugs*

VERY few therapeutic additions to medicine and surgery actually are new. Anticoagulant therapy joins the antibiotics, cardiac surgery, the revolutionary changes in anesthesia and blood and fluid replacement on a quantitative and qualitative basis as an elemental addition. Surgeons have been slow in accepting this measure and the new field that it opens. Internists are far ahead both in the control and application of the antithrombotic principle. This is understandable, as the surgeon must keep in mind a fresh operative wound. Sufficient experience has accumulated, however, to make this attitude no longer tenable. Certain fundamental controls are necessary, and when these are applied these drugs are not only safe but at times a lifesaving addition to the surgical armamentarium. These drugs are as safe as their user.

The term "anticoagulant drug," utilized for the past ten years, has become suggestive of hemorrhage to many surgeons. We purposely term these drugs *antithrombotic* to denote their therapeutic use rather than to describe the occasional accident which follows their misapplication.

These antithrombotic drugs prevent propagation of clots and possibly may dissolve some clots. Correctly utilized, this therapy prevents propagation of small clots—embolism—and therefore death. These drugs also will eliminate the necessity of surgical intervention in many patients. There can be no inflexible or definitive type of therapy. The treatment depends upon the stage and extent of the pathology and the response to therapy. The surgeon who decries all antithrombotic therapy probably is unskilled in its use and to cover such failure cries "Wolf!" Equally dangerous is the internist who knows that there is a soft clot present in the femoral or iliac vein but refuses to suggest that surgical ligation block that clot from extension to the heart or lung. Both of these extremists will have deaths which are preventable. In the early days of the use of these drugs the fresh surgical wound was placed first on the list of contraindications to antithrombotic drugs. This forced the surgeon to use operative measures. The failure of such a one-sided program has been reported. Over 1600 prophylactic ligations on postoperative patients in one hospital in a year resulted in many vascular cripples. Separating the medical from the surgical patients and treating only the former by anti

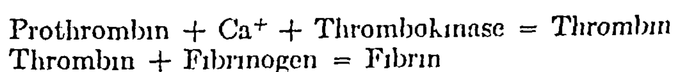
thrombotic drugs eliminated from such therapy a group which suffered often fatally from this complication We cannot plot a course in every case correctly, and some errors will occur If one will study and individualize the patient, know the drug and its action, and have a dependable laboratory, antithrombotic therapy in most surgical patients is safe and often lifesaving

Antithrombotic therapy originated with the discovery in 1916 by McLean[26] in Howell's laboratory of two phosphatides, cuorin and a heparphosphatide, which retarded clotting This heparphosphatide varied in its nitrogen-phosphorus ratio from the original Baskoff preparation Later Howell and Holt[18] isolated heparin in a crude form The pure substance is available due to the work of Charles and Scott, Jorpes and Bergstrom, and Schmitz and Fischer, according to the excellent summary of this substance's synthesis by Mason [25]

## ANTITHROMBOTIC DRUGS

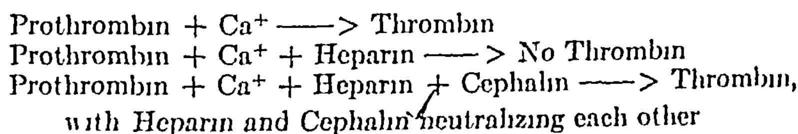
There are two main forms of antithrombotic drugs available today These are the injectable, rapid-acting ones, and those taken by mouth with a slower and more prolonged effect The former affects the bleeding and coagulation time The latter increases the prothrombin time Of the injectable substances, the one best known and standardized is heparin Examples of the second group are the coumarins· Dicumarol, Tromexan and the indanedione group

**Injectable Antithrombotic Substances.**—1 **Heparin** —While the synthesis of this drug was begun in 1916 in Howell's[18] laboratory, its purification was difficult enough to delay its availability for clinical use until 1933 It has a strong negative charge and its action depends upon the inactivation of thrombin It also acts as an antiprothrombin and may restrict platelet agglutination Adding this substance to Howell's original formula.



Prothrombin + $Ca^+$ + Thrombokinase = Thrombin
Thrombin + Fibrinogen = Fibrin

When heparin is administered



Prothrombin + $Ca^+$ ——> Thrombin
Prothrombin + $Ca^+$ + Heparin ——> No Thrombin
Prothrombin + $Ca^+$ + Heparin + Cephalin ——> Thrombin,
*with Heparin and Cephalin neutralizing each other*

Heparin complement is necessary for its action Heparin has a transitory action, 10 mg lasting forty minutes, 25 mg, one hundred and twenty minutes, and 50 mg only one hundred and eighty minutes, these figures being average for normal individuals

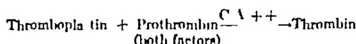
Howell theorized that heparin reacts with prothrombin to prevent its change to thrombin even in the presence of calcium It was his belief that in the action of heparin a substance, antiprothrombin, was developed which caused the prothrombin to become antithrombin

To understand the physiology of normal clot formation and how heparin interferes with this process, see formula above.

Whether heparin is a normal constituent of the blood or whether it is produced only when clotting occurs physiologically or pathologically is

disputed There is some inhibitory substance which maintains the normal fluidity of the blood and this is not extractable as heparin Another factor in the blood plasma which accelerates the activation of purified prothrombin by thromboplastin has been identified as Serum Ac-globulin which is formed from plasma Ac-globulin by the action of thrombin This appears to be the same as Factors V and VI described by Owren

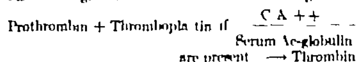
Thus a new equation evolves



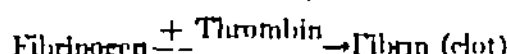
Thromboplastin + Prothrombin (both factors) —CA++→ Thrombin



Plasma Ac-globulin — Thrombin → Serum Ac-globulin



Prothrombin + Thromboplastin if CA++ Serum Ac-globulin are present → Thrombin



Fibrinogen + Thrombin → Fibrin (clot)

These theories are surgically not important Heparin when introduced into blood vessels will prevent intravascular clotting at the time of a surgical operation on those vessels and for a short time thereafter

*Action of Heparin* —Heparin is of low osmotic pressure high molecular weight and has a strong negative charge The anticoagulant effect apparently is due to the acid groups in heparin although the exact mechanism is confused It is quite certain that heparin prevents the action of thromboplastin on a quantity basis as sufficient heparin will inactivate thromboplastin Thromboplastin also inactivates heparin The tests of the action of these various substances are so varied that it is difficult to correlate them Heparin collects in the kidneys and is excreted through the urine 25 per cent of the dose appearing within an hour The fate of the rest is open to question The first clinical use of heparin in thrombosis was by Holmin[17] in 1937 who treated a patient with thrombosis in the central vein of the retina Heparin has been used extensively in the prophylaxis and therapy on various types of clotting since that time

Its use in coronary thrombosis was started hesitantly because in many cases of coronary thrombosis some of the coronary artery blockage is due to hemorrhage into an atheromatous plaque Since 37 per cent of patients with coronary occlusions produced embolisms anticoagulant therapy was considered justified The coronary disease patients treated successfully with anticoagulants now number over 3 000 [46]

*Heparin in Thrombosis* —Bauer s report from Sweden in 1946[1] in which he decreased the mortality of thrombosis from 17 per cent to 1 per cent by treating patients with heparin brought this treatment into vogue In his latest comparison of fatalities from thromboembolic disease Bauer reported a mortality rate of 1 3 per 10 000 admissions over nine years at the Mariestad Hospital in Sweden the therapy being intravenous heparin Control figures of mortality in ten other hospitals were 3 per 10 000 In those cases of diagnosed thrombosis with heparin therapy the mortality was 0 4 per cent. Bauer believes that only the intravenous injection is efficacious In this he is supported by Jorpes[20] and nearly all other Scandinavian investigators

*Advantages of Heparin*—The advantages of this drug can be stated simply It acts rapidly and quite consistently and can be given both by vein or intramuscularly

*Disadvantages of Heparin*—It is costly, difficult to control from the laboratory standpoint, and of short effectiveness

*Methods of Heparin Administration*—(A) *Continuous Intravenous Method*—The difficulty and inconvenience of this method, both for the patient and his attendants, have relegated it to the rare occasion where another method is impossible

TABLE 43—TYPES OF ANTITHROMBOTIC SUBSTANCES (1,170 PATIENTS)

INJECTABLE ANTITHROMBOTIC SUBSTANCES

| *No Given Injectable Drugs* | *Heparin 24 Hours* | *Heparin Over 24 Hours* | *Massive Heparin* | *Thromboeid* |
|---|---|---|---|---|
| 645 | 430 | 35 | 28 | 32 |

ORAL ANTITHROMBOTIC SUBSTANCES

| *No Given Oral Drugs* | *Dicumarol* | *Tromexan* | *Others* |
|---|---|---|---|
| 1130 | 758 | 360 | 12 |

AUTHOR'S SERIES[5,6]

(B) *Intermittent Intravenous Method*—This was the mode popularized by Bauer[1,2] in Sweden and has a place in the therapy, but due to the rapid rise and fall of the coagulation time under such a regimen, many fear an increased thrombosis may result Other forms of administration seem preferable

(C) *Injection of Heparin with a Slowly Absorbed Media*—This method was popularized with the use of the menstruum of Pitkin The inconsistency of the patient's reaction, the local pain and the occasional allergy caused us to discontinue this method although recognizing its value and place under certain circumstances Depo-heparin* sodium has similar properties with less reaction

(D) *Deep Subcutaneous Method*—This way of administration has proven effective in our hands Too rapid absorption or local bleeding responds to the use of ice bags and compression From 30 to 70 mg every three hours maintains the coagulation time and the therapeutic level in most patients The coagulation time (Lee-White method) should be checked before the administration of more drug

(E) *Regional Heparinization*—This method has some application. A cannula or plastic tube is tied in the vessel above the site where thrombosis is feared, *i e*, a point of vessel anastomosis The drug is then dripped into the area in a dilute form The danger of bleeding exists, when the wound in the vessel is kept open Its application is valuable at times in arterial surgery[14 21]

(F) *Massive Doses of Heparin*—We have used heparin** in 200 mg doses given at one time, deep in the subcutaneous tissues of the leg or arm

* Upjohn Co., Kalamazoo, Mich

** Supplied by Organon, Inc , Orange, N. J

by a fine needle Local bleeding is controlled by Ace type bandages and ice bags. A definite pattern of response follows with a rise in the coagulation time to as high as seventy minutes in four hours and then a gradual subsidence but continued therapeutic effect for twenty four hours In 25 patients so treated there was no bleeding This method with further modifications which will follow longer clinical trial has the advantage of the single injection and prolonged effect without the complications which occur when a form of heparin with a menstruum is given

(G) *Oral Heparin* —The use of heparin by mouth has been reported [19] We know of no confirmatory reports on its effect from United States' clinicians Wright reported no change in the clotting time as run by his laboratory after sublingual heparin was administered [45]

*Length of Time for Heparin* —If the oral drugs are begun at once heparin is used only until the prothrombin time is raised

2 **Other Injectable Anticoagulants** —Of the polysaccharide sulfuric acid derivatives so far synthetized the clinical results have failed to follow early enthusiastic claims

(A) Paritol (Polysulfuric Acid Ester of Polyanhydromannuronic Acid) —Paritol is an intravenous preparation which prolong clotting time between two and three times as long as heparin Its effect on the prothrombin time is minimal Reactions however occur and vary from swelling of the extremities to vascular collapse Some reaction occurred in 1 of every 10 patients This substance cannot be recommended until such reactions are minimized or eliminated

(B) Thrombocid, another polysaccharide sulfuric acid ester was used experimentally by our Clinic with others In a study of 32 patients with this substance the clotting time was prolonged definitely and on a predictable basis Two types of reaction were encountered In some patients a gastrointestinal upset varying from nausea and vomiting to diarrhea occurred In one-quarter of the patients an alopecia occurred in six weeks and in several of these it was a complete one In others it was a general loss and one patient forwarded a cigar box full of her hair to demonstrate the extent of the baldness Fortunately cessation of the use of the drug was followed by re-growth of hair Interestingly enough the hair which grew back was curly The possible beneficial cosmetic effects of this fact were not sufficient to make us interested in continuing the trial of this drug [24]

(C) Other similar polysaccharide substances have like complications

(D) Treburon is a derivative of polyhexuronic acid It is the sodium salt or sulfated polygalacturonic acid methyl ester methyl glycoside [19] It has one-third the potency of heparin and has not had sufficient trial for evaluation.

(E) Intravenous 4-OH Coumarin —A retardation or too rapid absorption of the oral antithrombotic drugs has led to difficulty in their control. Sometimes the drug passes through the alimentary tract without effect At other times the drug may be rapidly absorbed In an effort to overcome this inconstant factor a substance which can be injected directly into the blood stream has been reported by Link and used by Shapiro The sub-

stance is called Warfarin Sodium Derivative Its action is similar to Dicumarol and no side effects except hemorrhage have been noted in its limited trial The prothrombin time is lengthened in from twelve to twenty-four hours, and the effect has been reported to last for five days It has the obvious effect of 100 per cent absorption Liver damage was not noted[49] Its use has been limited

The dosage is 1 mg for each kg of body weight The minimum dosage is 50 mg and if the patient weighs over 80 kg. the dosage is 100 mg.

**Antithrombotic Drugs Which Can be Taken by Mouth.**—A **Dicumarol.**—3,3-,ethylene-bis (4-hydroxycoumarin) —The origin and synthesis of this substance is well-known to all and reads like a Disney fantasy The cattle that died of bleeding after eating spoiled clover played their part in the development of the coumarin group of antithrombotic drugs The safety of these drugs, when the fundamental precautions necessary for their use are fulfilled, has been established This fact is verified by figures from our own Clinic

*Advantages*—The oral route simplifies the administration The drug is widely available and can be administered for five cents a day

*Disadvantages*—This drug requires adequate laboratory control—which may restrict its use An accumulative action occurs at times The elimination of the drug must be regular and certain This requires a renal and liver system which is functioning satisfactorily All patients' response to the drug is not the same, and therefore the dosage must be individualized In general, however, the patient's response to the drug, once ascertained, can be relied upon in the presence of a normal liver and organs of elimination Contraindications to the use of the drug exist and will be given with the contraindications to all antithrombotics

*Dosage*—Usually 300 mg the first day and 200 mg the second day prolongs the prothrombin time to two times the normal on the third day Maintenance dosage averages 100 mg per day

B **Tromexan.**—3,3'-carboxymethylene bis (4-hydroxycoumarin) ethyl ester —This drug is one of the coumarin series and had extensive trial in Europe before its introduction into this country It is about one-fifth as strong as Dicumarol and takes action in eighteen to twenty-four hours The effect ceases twenty-four hours after administration is stopped[4, 7, 11, 12, 22, 33, 38, 43, 44]

*Advantages*—There is less tendency to hemorrhage with this substance It has the other advantages of oral administration. The earlier prolongation of the prothrombin time and the quicker return to normal once the drug has been discontinued is therapeutically advantageous. Its rapid elimination is due to a higher urinary excretion of the substance

*Disadvantages*—Some patients are resistant to Tromexan The drug is more expensive than Dicumarol It may cause hemorrhage, as may any of the other coumarin substances if not used cautiously

The contraindications will be considered with all of the drugs in this group

*Dosage*—This drug requires individualization In the average patient 1500 to 1800 mg will increase the prothrombin time to between twenty

and thirty seconds The initial dosage usually is 1200 to 1500 mg The maintenance dose is from 450 to 900 mg dependent upon the prothrombin time Wright noted a prolongation of prothrombin time consistently with 1500 to 1800 mg of Tromexan [44] In an analysis of our figures, we found a larger amount necessary We gave an initial dose of 1200 mg followed by 900 mg the second day to the patients who had thrombosis of either venous or arterial nature On the average it took us 3 2 days to reach a high therapeutic level (twenty five to thirty seconds) The dosage necessary to reach that level varied from 1200 mg to 4800 mg and averaged 2 760 mg [24] This figure while higher than others reported indicates that more of the drug is necessary than we formerly believed or that thrombotic patients develop a resistance to it The relative safety of the drug is demonstrated by the fact that we have carried patients on this drug on an ambulatory basis for over 3 years with no serious hemorrhages These were clinic patients on the lower West Side of New York where often one cannot rely too much on the patient s reports [26]

C **Phenylindanedione** —Danilone—PID This oral drug is one of the indanedione derivatives and acts by lowering the prothrombin time

*Advantages* —Rapid action and low cost

*Disadvantages* —The main disadvantage in its use is that five to ten per cent of the patients are resistant to the drug In these patients necessary and valuable time may be lost before this resistance is demonstrated Previous toxic effects which included hematuria and kidney blockage have been eliminated The drug acts more actively than Dicumarol The therapeutic level of approximately 30 per cent of normal prothrombin activity will be reached by the majority of patients within thirty-six hours The bleeding tendency is not counteracted by vitamin K [44]

*Dosage* —Two hundred to 300 mg for patients over 150 pounds one-half of the dose each in the morning and at night has been advised by Blaustein.[14]

D **Cyclocoumarol** —Compound 63—Methopyranorm (4-Hydroxycoumarin) This substance has been known as Number 63 or BL-5 has the formula 2 methyl 2 methoxy-4 phenyl-5-oxodihydropyrano [3 2-C]-[1] benzopyran and was developed in Link s laboratory by Ikawa This drug has long action Therapeutic levels have been maintained by giving the drug every two to five days The advantage according to Hansen *et al* is less fluctuation in prothrombin time [15] The disadvantages are that the effect cannot be terminated when desired and that vitamin K does not counteract the drug to any appreciable amount. Vitamin K 1 in doses of 200 to 500 mg has stopped bleeding at least temporarily

*Dosage* —The drug has been used in a single dosage of 2 mg per kg of body weight. The average dose is 125 to 150 mg The therapeutic effect is reached in twenty four to forty-eight hours

E **Combined Coumarin Antithrombotic Therapy** —It has been felt by all observers that the ideal antithrombotic substance was not yet synthetized It may be that the final substance which will achieve the ideal result—elimination of thrombotic episodes without hemorrhage—may be a combination of the substances

The combination of Tromexan (ethyl biscoumacetate), which acts rapidly, with Cyclocumarol (cumopyrin which acts only after 48 hours but which acts more stable thereafter) has been advocated

*Dosage* —First day: Tromexan (ethyl biscoumacetate) 1200 mg Cyclocumarol 50 mg Subsequent doses of Tromexan 300 to 600 mg Subsequent doses of Cyclocumarol, depending on the prothrombin time, 25 to 50 mg

**Lipid Substances Reducing Blood Clotting.**—These substances are mentioned for their future possibilities and not for their present therapeutic effects

Some of the lipids have an inhibiting effect on thromboplastin [31,32,35]. These substances are prepared from 2 per cent emulsion of dried beef brain, acetone, and also from rabbit lung and the soybean [42] The effect is by action on Factor V, or the labile factor

**Derivatives of Alphatocopherol.**—The preparation of vitamin E has been used for nearly every disease and in every therapeutic way Prothrombinopenic effects have been claimed for it Experiments reported by Ochsner,[25] that alphatocopherol plus calcium develops an active antithrombin, have not been confirmed by others We were interested and used large quantities of alphatocopherol, with calcium gluconate, postoperatively We found no evidence of the antithrombotic effect In our series there were 3 patients who developed a postoperative phlebitis, compared with 2 in a control group We have tried it also on our ambulatory thrombotic patients with equivocal results, the episodes of thrombosis following approximately their usual incidence McLachlin *et al* [27] reported a complete lack of correlation as to prediction of venous thrombosis by the antithrombin test during life and the presence or absence of venous thrombi at autopsy Wright[17] and others[33,34] found no anticoagulant effect from this substance

**Length of Time of Oral Drugs.**—If these drugs are utilized, they should be continued at a therapeutic level for at least three weeks In exceptional cases, such as blood vessel surgery, one needs to use them only until it is certain that blood is passing the suture line without stagnation and clotting, for example, three days In the chronic thrombotic or migratory thrombotic patients, in those who have had arterial embolism, and probably in most of the coronary cases, antithrombotic therapy should be continued indefinitely.

## INDICATIONS FOR ANTITHROMBOTIC THERAPY

1. Venous thrombosis, either of the inflammatory or bland type
2. Complications of venous thrombosis such as extension or pulmonary embolism
3. Arteriosclerosis with incipient or active gangrene
4. Arterial thrombosis of the traumatic or nontraumatic type
5. Serious injuries to blood vessels
6. After operations on blood vessels to prevent thrombosis
7. Coronary arterial disease
8. Restriction of the circulation due to the exposure to cold or wet
9. Mesenteric thrombosis of either an arterial or venous nature

10 Acute arterial occlusions which may be due to trauma infection muscle or bone pressure arterial embolism or to exposure (*i e* frostbite immersion foot or trench foot)

11 Postoperative and postpartum in selected instances to prevent thrombosis or embolism where there is a history of previous episodes or where other lesions exist which would make their occurrence likely

## CONTRAINDICATIONS FOR ANTITHROMBOTIC THERAPY

1 Conditions raising the prothrombin level
2 Vitamin K deficiency
3 Liver disease
4 Advanced kidney disease
5 Late pregnancy
6 Vitamin C deficiency
7 Pulmonary tuberculosis with hemorrhage
8 Blood dyscrasias
9 Gastrointestinal bleeding *i e* peptic ulcer
10 Fresh wounds—not all contraindicated
  *a* Open wounds *i e* gunshot except extremities
  *b* Operative wounds especially
    (1) of the brain
    (2) of the lung
    (3) where there has been inadequate hemostasis
    (4) most draining wounds
    (5) after operations for jaundice
11 Subacute bacterial endocarditis

Note These indications and contraindications generally apply but it is important that the therapy for each patient be individualized

## LONG TERM ANTITHROMBOTIC THERAPY

Some patients with cardiac arterial or venous diseases are in constant danger from clotting The patient who has auricular fibrillation may develop arterial embolism In a like manner the arteriosclerotic may break off a plaque or clot as the blood stream slows down in the occluded vessel Inflammatory venous clotting may migrate Many patients develop re-

TABLE 44 —USE OF ANTITHROMBOTIC DRUGS ON GENERAL SURGICAL PATIENTS[34]

| *Number Patients* | *Number Hemorrhages* | *Cause of Bleeding* | *Per Cent Hemorrhages* | *Per Cent Hemorrhages at Therapeutic Level* |
|---|---|---|---|---|
| 494 | 9 | Overdose 6<br>Insecure wound 3 | 1.8 | 0 1 |

peated attacks of mesenteric thrombosis The individual and family clotter have been discussed Such patients are in danger of the loss of life or parts of the body These patients may be put on anticoagulant therapy and carried indefinitely We have successfully used such a program at the

Vascular Clinic at St Vincent's Hospital on a large number of patients for three years Prior to that time we had so treated individual patients for over eight years Among these were patients who had had mesenteric thrombosis, arterial emboli, pulmonary emboli and migrating thrombitis

These patients are not hospitalized, but their response to the drug is plotted for a period of two weeks Thereafter their prothrombin time is taken twice a week and the dosage of the drug computed from this test At a later date the prothrombin test is done weekly and then bi-weekly. Where there has been *no serious liver, kidney or hematogenous disease* there have been no accidents One patient bled after a dental extraction To prevent accidents, the following direction slip is given to each patient

THE ST VINCENT'S HOSPITAL OF THE CITY OF NEW YORK
VASCULAR CLINIC—SPECIAL DRUG THERAPY

*Important* The medicine you are taking will help you It is necessary to follow each direction and telephone or come in if there is any *unusual* development

1 To report for blood tests at 9 A M on days appointed
2 Take tablets exactly as prescribed by doctor after each blood test
3 Do not have any type of surgery done (for example, extraction of teeth) without first consulting your doctor in Vascular Clinic

*Watch for*

1 Blood in the urine and stools (Urine becomes "smoky" in appearance or red )
2 Bleeding areas under the skin They appear like a black eye
3 Bleeding from the gums, nose or any other part of the body
4. Prolonged bleeding from cuts or bruises

Tromexan has worked best for this therapy in our hands

The clinic patients for the most part are poorly educated and do not understand a great deal of English We believe that any group can be treated safely on antithrombotic therapy with careful observation and follow-up It is our practice to advise such therapy in those patients who are in serious danger of clotting or embolism but *only* when laboratory and clinic control is available and used

## MANAGEMENT OF HEMORRHAGE DUE TO ANTITHROMBOTIC DRUGS

PROPHYLAXIS —The best treatment for hemorrhage from antithrombotic drugs is to prevent it The administrator of the drug must have skill and knowledge The surgeon must know the drug he uses, its action and its dangers He must also be certain that the laboratory reports will be performed consistently and accurately The status of the patient and his ability to eliminate the drug must be ascertained from time to time The contraindications must be recognized unless the danger of thrombosis is greater than that of hemorrhage In the early days of anesthesia, many patients died An overdose of spinal anesthesia or pentothal will kill as quickly as a bullet The failure to relieve a pneumothorax is as fatal as potassium cyanide Yet, we do not hesitate to use anesthesias, to open pleuras, and even to put our hands and instruments within the human heart In a similar manner, diligence makes these drugs safe There is no half-way measure The patient cannot be turned over to an inexperienced interne, and many

patients have suffered from the long weekend with the laboratory closed These drugs know of no holiday If one is not prepared or equipped to exercise due caution the drug should not be administered If the above criteria have been fulfilled the drugs will be safe, helpful and often life-saving

**Hemorrhage from the Injectable Drugs** —Heparin does not change or dilate blood vessels Local hemorrhage or hemorrhage from a wound is possible Bleeding from the mucous membranes has occurred The treatment should be (a) Stop the drug (b) For a local hematoma apply pressure and ice bags (c) *Whole fresh blood* should be given and continued as often as needed The blood should not be more than two days old (d) Protamine sulfate may be given intramuscularly (50 mg of protamine sulfate in 5 cc aqueous solution every six hours) In severe hemorrhage 150 mg can be given intravenously in one hour (e) Toluidine blue a vaso-dye counteracts the heparin action Two mg per kg of body weight is given intravenously

**Hemorrhage Due to the Oral Antithrombotic Drugs** —(A) Discontinue the drug

(B) Local hemostasis The surgical hemostasis must be secure if anti thrombotics are to be used It is wise to increase the pressure at the operative site Packing may be required

(C) Whole fresh blood should be given by transfusion Bank blood is not as effective Such blood supplies prothrombin to the deficient plasma In from two to six hours this is used up and the prothrombin time rises again The treatments must be continuous and continued until the bleeding has stopped both clinically and chemically

(D) Vitamin K active compounds Vitamin K K-1 and K 2 are present naturally in the body Their action is due to basic naphthoquinones If one uses a synthetic naphthoquinone one must give much larger doses (64 to 75 mg every four hours) Vitamin K should be given intravenously Synthetic vitamin K acts only in eighteen to thirty-six hours and therefore is not reliable Vitamin K-1 is superior to vitamin K Synthetic vitamin K can supply 40 per cent of the activity of K plus K-1 according to Quick [37] Vitamin K 1 is supplied in an emulsion for intravenous injection as a 5 per cent solution It acts in fifteen minutes and reverses the prothrombin time range in four to twelve hours (usually three to six hours) Vitamin K 1 oxide has had comparable effects but there is a latent period of several hours before it acts In serious cases it may be given at the same time intravenously [13,31,46]

It is important to emphasize that the vitamin K substance must be continued and be combined with fresh whole blood These drugs will stop the hemorrhage Most accidents have happened due to the lack of giving sufficient doses not continuing them long enough and not using the fresh blood in combination

## SYMPATHETIC NERVE BLOCKS AND ANTI-THROMBOTIC DRUGS

The value of sympathetic nerve blocks and antithrombotic drugs in many forms of thrombosis or prophylactically in arterial occlusions is

well-known [35b,35d] The indications for perivertebral blocks often are identical with those for antithrombotic therapy Some authors have reported on serious and fatal injuries from the combination of these two procedures [10,23,29]

For many years we have combined these methods of therapy Of the 630 patients who have been treated for thrombosis, 566 have had sympathetic nerve blocks in combination with antithrombotic drugs These patients had from 2 to 15 blocks each, with an average of 4 We have performed, therefore, 2,152 sympathetic nerve blocks on patients receiving therapeutic antithrombotic drugs There has not been a single hemorrhage in this group During the same time, we have been consulted for three massive retroperitoneal hemorrhages occurring after blocks where there was no antithrombotic therapy being administered We feel strongly that hemorrhage after such a procedure is technical in nature Our point is not to emphasize that the two therapies should be given together, necessarily, but it is to indicate that with due care, hemorrhage does not occur with the use of antithrombotic drugs even when one is introducing 3 one-half inch needles to their full length near major vessels

TABLE 45 —CO-ADMINISTRATION OF SYMPATHETIC NERVE BLOCKS AND ANTITHROMBOTIC THERAPY—VASCULAR PATIENTS

| *Number Patients* | *Number Blocks* | *Average No Blocks* | *Hemorrhages* |
|---|---|---|---|
| 566 | 2152 | 3 8 | 0 |

*Safety of the Antithrombotic Drugs* —As surgeons we should use any measure that will help our patients to get well We must know that such an addition to therapy is safe Our studies indicate that the use of these antithrombotic drugs is safe

In the last seven years, from 1945 to 1952, we have treated 630 patients with arterial or venous thrombosis with antithrombotic drugs During the same period we have treated 494 surgical patients for some complication of a thrombotic nature or prophylactically where thrombosis was to be expected Of this total number of 1,113 patients, 795 were treated with Dicumarol and 318 had Tromexan administered to them The majority (75 per cent) were given heparin for the first twenty-four hours One group of 32 were given an injectable heparin-like substance, Thrombocid, exclusively Another group of 25 patients had the massive dose heparin treatment exclusively In this large group there have been 9 hemorrhages, an incidence of 0 8 per cent This is lower than the over-all hemorrhage rate with anticoagulants The average hemorrhage rate in postoperative patients including minor ones is 6 per cent During the same period there were 8 massive hemorrhages in general surgical patients without the use of antithrombotic drugs

Analyzing the bleeding patients who had antithrombotic drugs, one died after a splenorenal shunt for esophageal varices The autopsy showed many ulcers in the varices area and bleeding from any one of them was to be expected despite the therapy The possibility that the antithrombotic drugs contributed to the picture exists but is questionable

Three patients bled from recent wounds—1 after an inferior vena cava ligation (patient had had heparin) 1 after an extraction of a tooth and 1 after a hysterectomy These were all controlled with blood transfusions One patient on ambulatory Tromexan developed subcutaneous bleeding and another had a subconjunctival hemorrhage These were both treated only by the cessation of the drug One of our investigators developed kidney bleeding Of these hemorrhages 7 occurred from overdosage The other two namely the tooth extraction and the esophageal varices patient who died bled at therapeutic levels and therefore the part that these substances played in the bleeding is questionable The patient with esophageal varices bled from ulcerated varices with a prothrombin time of eighteen seconds

Our Clinic has used these drugs not sporadically but regularly and not for months but for years We have taken all the precautions necessary we believe for safety and in our hands the drugs have been used safely It is our contention that if these drugs can be used in one surgical clinic where all the safeguards necessary are taken they can be used in any clinic similarly equipped provided that the same safety measures are instituted These findings are supported by those of Smith *et al* [41] who administered Dicumarol to over 3 078 females over 35 who had major surgery with no deaths and no serious bleeding

*Notes on the Future*—Certain future possibilities are being investigated and others exist These are

1 Counteracting the vitamin K deficiency during antibiotic therapy with vitamin K injections

2. The addition of vitamin K or whole blood to antithrombotic therapy in those patients in whom bleeding is suspected Thus the antithrombotic substances would be present with a safeguard against bleeding

3 Broadening of the ambulatory antithrombotic therapy to include all patients with malignancy or other thrombotic tendencies The use of these drugs for lifetime in those needing them is inevitable as soon as other safety factors have been devised

4. The dissolution of early and late clots as has already been shown to be possible in veins using some of the kinase substances has many other facets. We are working at the present time on the dissolution of artery thrombosis with such substances

## PROTHROMBIN TEST—PROTHROMBIN DETERMINATION

**Method of Reporting Prothrombin Determination.**—The prothrombin test has been reported in *seconds* and in percentage of activity The former is the registration of the actual time necessary for the fibrin clot to form It is the end point at which the clot is stable enough to permit it to be moved by a stir leaving a clear area Using whole plasma this time normally is from thirteen to seventeen seconds This can be more accurately recorded with an electric timer If the 12 5 per cent dilution is used the normal time is thirty five to forty two seconds The test should be reported in relation to a normal for that reagent used that day In reporting the prothrombin *in percentage of activity* it is necessary to employ a dilution

curve Such a curve should be based on many determinations It is better therefore for the test to be reported clinically in seconds If the percentage of activity method is used the clinician should ascertain the exact method used to calculate it, what curves are used, etc. This latter method appears to expose the patient to more hazards

**Plasma Prothrombin Test.**—This test has been well-standardized by the accumulative efforts of the many investigators The Link and Shapiro Modification of the Quick test is the one used by nearly all hospitals Its accuracy requires the experienced technician In most laboratories such technicians perform no other tests The technic as such is not detailed, as it is available in all modern laboratories and medical vascular texts [1a,35 35a,40,46,47]

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Chapter

# 36

# THE SURGICAL MANAGEMENT OF THE POST-THROMBOTIC SYNDROME

## *Ligation for the Enlarged Veins Treatment of Ulcers Selection of Patients for Sympathectomy*

THE management of the acute stages of pathologic venous clotting has been considered on pages 617 and 627 As the result of such clotting whether it be inflammatory primarily or not residual changes occur in a certain percentage of patients These patients are now classified by their symptoms pathology complications and disability into a well-established syndrome It has been called the postphlebitic postthrombotic postedematous post varicose syndrome and by other descriptive names We typify all of these as the postthrombotic syndrome and subdivide it further depending upon the complications

In approximately 25 per cent of patients with acute thrombosis spontaneous resolution is complete [11] In 3 out of 4 however varying degrees and types of disability remain The pathogenesis of the syndrome will be reviewed briefly to make an understanding of the complications more simple

**Pathogenesis.**—In *thrombitis* the inflammation is primary and the clot of secondary origin The inflammation causes spasm in the affected vessel and in the collateral veins In the rare patient this spasm factor may be so great as to cause arteriospasm and gangrene has occurred (see pages 298 to 300) This spasm is initiated at the site of the original lesion By a reflex synapse (neurogenic theory) or by the activation of some circulating substance which causes the vessel contraction (chemical theory) the symptoms develop These are inflammation in the involved vessel swelling pain and spasm of the involved veins and other collateral vessels [7,8]

In *thrombosis* the clot at first is not inflamed and propagates along the vein wall. When it occludes that vein the pressure behind it develops rapidly Embolism may occur at such a time If it does not back pressure edema and later, all the other signs of inflammation result Thus despite the variation of the original cause the end picture is the same If the recanalization occurs early and is complete and if the pathology has not been too extensive, the leg may return to normal or near normal status (25 per cent) If many veins are involved primarily or by spasm edema will be the dominant picture The percentage of edematous legs increases if adequate support for a sufficient length of time was neglected The super-

ficial femoral vein most often is involved Its three or four valves are destroyed by the inflammation The vein's ability to return blood, even after recanalization, is impaired or absent The blood flow is reversed and in these patients pigmentation deposits, skin changes, secondary eczema or fungus infections occur Ulcers follow a minor trauma In our Clinic's experience, these postthrombotic ulcers are present in one out of every three such patients [9,11,16a]

In addition, in most of these patients the saphenous system has been overused, its valves have failed, and its pathology is similar to that in simple varicose veins In roughly 40 per cent, edema is a prominent symptom and this covers many dilated incompetent veins Spasm continues as a factor and the symptoms detailed below occur in varying degrees Many of the complaints are similar to those of causalgia Often such patients have become neurotic as they have failed to obtain relief from the many physicians they have consulted The multiplicity of their complaints, their bizarre nature, and the apparent innocuous clinical findings have contributed to this mental status

In a few patients, the inflammation recurs time after time in the affected leg In a minority (possibly 3 per cent) thrombitis develops in other parts of the body and becomes a migratory type Many such patients with thrombitis migraines have malignancy, and a diligent search for carcinoma, sarcoma or Hodgkin's disease should be made

**Symptoms of the Postthrombotic Syndrome.**—The symptoms of the posthrombotic syndrome are as follows

1 *Swelling* —The swelling may or may not be severe, but it is consistently present In some, it occurs because the patient has not adequately supported the part for a sufficient length of time The patient constantly is aware of the swelling It is greater on dependency and always more apparent at night In certain individuals with good support, it disappears after six months, but in some it remains as a residual permanent swelling It is most severe if the thrombosis involved both the deep and superficial venous systems

2 *Heaviness* —The patient complains that there is a heavy, loggy feeling in the limb At times it is described as a leg they have to drag along after them The limbs have a wooden feeling in them, and some of the patients even develop a limp from this heaviness

3 *Pain* —Pain is a subjective symptom and therefore depends upon the patient's reaction to it The pain may be present only when the leg is full or swollen, but in many it is present all the time and is particularly aggravated at night This pain is described by these patients as a constant ache with electric shock-like feeling of pain down the leg The patient has difficulty in localizing the painful areas, but in general, the pain is in the calf, at times along the medial side of the thigh, frequently at the ankle, and in many in the buttocks and hips.

4 *Paresthesia* —With this pain, there is a paresthesia Paresthesias are described as lightning-like aches and pains Frequently, patients describe the feeling as that of worms crawling on the leg Many of these patients feel as if there is a bubbling or gurgling of fluid up and down the leg When the patient stands on the foot, he believes blood or some solution rushes to his limb

5 *Hyperesthesia* —Patients with the postthrombotic syndrome become extremely sensitive about the affected part and are like the patients with causalgia The hyperesthesia becomes the predominant symptom Many of our patients had discontinued wearing girdles belts and garters or any type of pressure support because of this sensitivity Some of these individuals will place the hyperesthetic leg behind them when they sit down for fear some slight trauma will disturb it Often they have devised some sort of a cage at home to keep the bedclothes off the part

6 *Anesthesia* —Areas of anesthesia are not unusual and this numbness at times is most alarming to patients They feel that their circulation is failing and it is for this reason that many of them consult their physicians

7 *Causalgia* —Many of these patients develop a true causalgia similar to that seen after a trauma to the limb This is like the causalgia seen in World War II in which the degree of complaint of pain and distress by the patient was in no way comparable to the injury received For many months in the early part of the war these patients with causalgia were not treated satisfactorily The degree of disability was far beyond the clinical findings. They had a great fear of any handling or manipulation of the part and finally withdrew into themselves When sympathectomy was first performed for these patients a great many of those operated on were already in psychiatric wards Court martial has followed refusal of such a patient to return to active duty or to a firing line after a relatively minor injury The postthrombotic causalgia may be as severe

8 *Skin Changes* —The skin becomes shiny extremely cold in most instances with some inconsistent areas of warmth There is atrophy of the limb due to disuse The part tapers joints stiffen and tips of the toes become blue

9 *Mental Changes* —These patients as already mentioned develop definite psychopathic personalities as the result of this syndrome For example we have seen patients who develop a postthrombotic syndrome after childbirth who decide never to have more children These instances often have been described by obstetricians It is usual to fear whatever caused the thrombosis This fear has been aggravated by doctor after doctor telling them that there was nothing to be done for it They become depressed about the problem They begin to develop a feeling of depression and hopelessness Belittling their complaints increases this feeling Compensable injuries may complicate the mental side of the picture

10 *Ulceration* —Ulceration and other complications have been discussed in the chapter on Pathologic Venous Clotting (See page 607 ) One-third of this group of patients have ulcers The ulcer begins about one year after the phlebitis[4,14] This is the time when the superficial femoral vein is trying to recanalize itself The pathologic picture therefore is similar to that of varicose veins with venous back pressure with the exception that the venous back pressure is not only in the dilated saphenous vein system but also in the superficial femoral system

**Pathology of the Postthrombotic Syndrome** —The pathologic picture is not too well-known because these patients do not die of their lesion the leg is not amputated and therefore the pathologic status much like that in causalgia, cannot be studied We do know that the valves of the superficial

femoral vein no longer function after thrombosis A probe or vein stripper passes retrogradely through such a vein without obstruction This vein becomes tube-like In the upright position there is a back pressure

With this stasis there is venous failure in the part drained by the superficial femoral vein Edema, pigment and dermatitis develop similarly as they do in saphenous vein failure, but the changes are more extensive Ulceration is a complication in approximately 30 per cent These ulcers may be multiple and extensive and most resistant to healing The ulcers may be on the internal or external portion of the leg and they may be serpentiginous in form The ulcers are characteristically painful, indolent and secondarily infected They do not respond to simple measures such as support In addition, the saphenous system, on which has been thrown an extra load, becomes incompetent, with failure and all of the pathologic changes which follow varicose veins The resistance to therapy is understood when the complexity of the pathology is studied There may be such a change in the venous return from the part that innumerable inadequate small venous channels are developed

The part that spasm plays is well-known, and this spasm may be on both the arterial and venous sides (Fig. 146) This spasm is reactivated by use of the limb or pressure on some area, such as in the calf, when the patient lies in bed That there may be secondary nerve changes on a reflex basis is without question because of the similar appearance that these patients develop to those with true traumatic causalgia

Atrophy of the limb is not unusual In some, there seems to be a relationship to atrophy of Sudeck's type The bone may show osteoporosis and calcium absorption A layer of edema between the skin and fascia displaces fat and all the superficial vessels and nerves The circulation to the part may be impaired by this pressure

**Surgical Treatment of the Postthrombotic Syndrome.**—A **Prophylactic Treatment.**—The edema, pain, and other symptoms (see page 668) which follow thrombitis or thrombosis often can be controlled if the surgeon institutes treatment In our experience, the satisfactory handling of the inflammatory type lesion is as follows

1 *Sympathetic Nerve Blocks* —Sympathetic nerve blocks should be performed with 5 cc of 2 per cent procaine injected into the four lumbar ganglia For the technic, see page 494 These nerve blocks should be repeated as often as is necessary Rarely, an anesthetic in oil may replace the procaine if a longer action is desired Where repeated sympathetic blocks over a period of hours or days are contemplated, a plastic catheter may be inserted This is inserted through the needle, the latter being withdrawn and the tube left *in situ*, plugged and in a sterile dressing No untoward results have followed this technic and it has been employed many hundred times [10]

The use of alcohol in an effort to produce a permanent or prolonged effect has been discontinued. The severe reaction to the alcohol or the alcohol neuritis which may develop with the pain, and at times atrophy of the limb, rules out the use of alcohol except in selected instances (See also page 498 )

2 *Elevation of the Part* —Elevation of the part is necessary to aid Nature's efforts to solve the problem of the venous return This elevation includes raising the foot of the bed and keeping the feet raised when the patient is seated The importance of the elevation cannot be overstressed This means that the part must be above the *heart* level and not merely resting on a low footstool During the day it may be necessary to raise the part many times and this should be done every time edema develops Elevation of this type for a period of twenty to thirty minutes will eliminate the swelling

3 *Warm Packs* —Warm packs are applied at the same time the part is elevated These warm packs have replaced cold packs The warm packs are applied to aid in overcoming the spasm of the collateral vessels and to relieve the pain To do this it is necessary that the packs be kept on for a long time The temperature should be controlled and the skin protected

The technic of applying warm packs is simple but it must be explained and emphasized to the patient or the nursing service in order that it be performed correctly

*Vascular Packs —Technic* —The packs should be applied as follows The skin of the extremity is protected by a thick layer of lanolin or petrolatum to prevent its maceration The part is then wrapped in two thicknesses of warm wet turkish towels Hot water bag in which the temperature has been carefully ascertained to be between 88 and 90 F are then placed on top of the wet towels and the entire extremity is wrapped in a large loose rubber sheeting or heavy blanket The wet towels will transmit the heat from the hot water bags throughout the extremity the heat being maintained by the external covering The foot should be included in the pack as otherwise it acts like a chimney and permits the escape of the heat Ordinarily the temperature will be maintained for one and a half to three hours in this way and the hot water bag can then be replaced The pack is used from three to eight hours a day During this time the patient should not be immobile but should move actively even in the pack.

4 *Potassium Permanganate Foot Soaks* —Potassium permanganate foot soaks should be used to control the associated fungus infection which is nearly always present Normally, a solution of 1 10 000 to 1 30 000 used twenty minutes every one or two days is sufficient for this purpose The solution should be deep enough in the tub to cover all irritated areas and any points of evident dermatitis on the feet or legs In applying these soaks small cotton pads placed between the toes near their tips will allow the solution to get into the crevices at the base of the toes where the fungus is prevalent.

5 *Saline Soaks* —A saline soak or a Sitz bath is utilized twice a day to provide warmth and localize any infection Saline is an innocuous solution Its use twice a day has the advantage of a wet dressing without the inevitable cooling and maceration which accompanies a continuous wet dressing

6 *Antibiotic Therapy* —In those with a marked degree of inflammation antibiotic therapy is indicated A culture of the lesion should be made and the sensitivity of the organism to the various antibiotics tested The

correct antibiotic for the organism then is selected If the growth is mixed, one or more of the antibiotics are used empirically Certain of these lesions suppurate and break down It is in an effort to control this part of the picture that antibiotic therapy is used

7 *Antithrombotic Therapy* —The prophylactic treatment also includes administration of the antithrombotic substances Heparin is given as detailed on pages 654 to 655 One of the oral antithrombotics is started at the same time and the heparin is discontinued when the prothrombin time is elevated

8 *Anticoagulants and Sympathetic Nerve Blocks* —In many clinics, the simultaneous use of sympathetic nerve blocks and antithrombotics is never performed It is our feeling that hemorrhage from sympathetic blocks even when antithrombotic therapy is in use is due to technical failure We have used the two simultaneously in over 2000 instances without hemorrhage During this same interval we have seen 4 severe hemorrhages from



FIG 208 —Pressure legging for leg support Pressure is applied by a bulb pump This legging is of value in venous stasis (Courtesy Dr W J M Scott, Rochester, N Y )

sympathetic nerve blocks in which there was no coexistent antithrombotic therapy These two therapies are considered important complementaries to each other This antithrombotic substance is carried on for approximately fifteen to twenty-two days For details, see the chapter on Antithrombotic Therapy, page 651

9 *Active Motion* —During this time, active motion is continued The limbs are kept moving, at first in bed, as long as there is inflammation present, and subsequently by graded walking, the foot being supported from the toes to knee with an Ace bandage when the patient is out of bed

10 *Support* —Thereafter, the limb must be supported when the patient is up At first, Ace-type bandages supply satisfactory support These must be adjusted several times a day to accommodate the reduction in size with walking After a static reduction stage is reached, elastic stockings can be worn to maintain this reduction This support should be continued for six to twelve months

inflammation, a demonstration of valve failure and proof of the adequacy of the femoral profunda system (see pages 676 to 677 for technic) Venograms are of importance in such selections.

## SYMPATHECTOMY FOR CAUSALGIA IN THE POSTTHROMBOTIC SYNDROME

Many of these patients, however, develop chronic recurrent symptoms with marked disability These symptoms are consistent and similar, and the patients may be classed as having the postthrombotic syndrome.

The excellent results which followed the treatment of acute inflammatory thrombitis with sympathetic nerve blocks have been observed for many years Often, after sympathetic nerve blocks, it has been astounding to see the clinical reaction of the patient The edema and pain frequently disappear, as if by magic, as does the inflammation [5,7,8]

Many years ago, we performed sympathetic nerve blocks on many of these patients in the later stages with excellent temporary results, but they later returned to the same status as before The possibility of permanent interruption of the sympathetics was considered many times Sympathectomy then was tried [9,13,15,16] This operation has been performed in our Clinic 165 times for the postthrombotic syndrome No untoward effects from the procedure have been noted and most of the patients have had marked relief of symptoms

In these 165 patients there has been a reduction of edema in two-thirds and, more important, the symptoms of heaviness, paresthesia, anesthesia, and coldness have been relieved. Rehabilitation in many patients to a better status of health has been possible It is important to explain to the patient with the postthrombotic syndrome that some swelling may persist for a long time or even permanently and that support to the limb may be required when there is to be overactivity and standing

Sympathectomy is not a cure-all for these conditions It is not like taking out an appendix for an appendicitis The patient does not have a normal circulatory status in the limb Certain changes have occurred which are definite and irreversible, and the patient, his family, and doctor must be so informed The surgeon's efforts in these cases are to make the patient more comfortable, to reduce the edema as far as it is possible, and to relieve as much as we can the causalgic type of distress of which these patients complain Sympathectomy therefore is of great value in the treatment of selected patients with the postthrombotic syndrome

**Selection of Patients for Sympathectomy.**—In the selection of patients for sympathectomy, certain criteria can be established The patients should be those who have continuing symptoms despite good conservative therapy Those who will respond best are the patients who have cold legs, who complain of heaviness, paresthesia and anesthesia, who have developed a marked mental reaction to the condition and who are willing to support the limb thereafter Nerve blocks will help select those patients who are most likely to respond to sympathectomy

**Sympathectomy for the Postthrombotic Syndrome**.—Part of the enthusiasm for the sympathectomy in the treatment of the postthrombotic syn-

drome is due to the fact that we have found it a simple operative procedure to perform. The operation of lumbar sympathectomy from the posterior approach is not only technically difficult and time-consuming but is traumatizing and at times followed by many complications. The anterolateral retroperitoneal approach is simple and will be the one of choice of most surgeons who will try it.[illegible]

**Operative Technic.**—Spinal anesthesia is used. This technic has been presented thoroughly on pages 507 to 510. It includes positioning and certain operative points which will not be needlessly repeated. The only technical variation in the sympathectomy for the postthrombotic syndrome is that many more collateral veins may be encountered.

## SURGICAL MANAGEMENT OF THROMBOTIC ULCERS

**Pathogenesis of Postthrombotic Ulcers.**—The ulcers which result after a thrombosis in the saphenous system usually are similar to simple varicose ulcers and respond to therapy for the varicose ulcer (see pages 776 to 781). Where the ulcer follows inflammation of the deep veins (usually the superficial femoral), a different type of pathology exists. Such ulcers cannot be healed and kept healed by elimination of a coexistent inadequate saphenous system alone.

When the superficial femoral vein has been inflamed and fibrosed its valves are destroyed and do not function satisfactorily again. In approximately a year this vein begins to recanalize.[4] There may be one or several openings in it but there is no valve function to guard against the back pressure. The femoral vein becomes a tube and when the patient is upright a back flow occurs in this system which is similar to that which occurs in an incompetent saphenous system. The area drained by this vein becomes suffused; there is edema, pigmentation, unsatisfactory oxygenation—all of these factors being prerequisites for the ulceration. A mild or unnoticed trauma may cause a skin break. Scratching or edema pressure may initiate the ulcer. At times no excitant cause is recognized. If one introduces a stripper into a feeding vein above such an ulcer, this stripper often will be found directly in the femoral vein.

**Femoral Vein Ligation.**—The history and physical examinations will determine if the superficial femoral vein was inflamed and is therefore the cause for the ulcer. This can be further proven by a common femoral venogram with contrast dye. With the patient standing, the incompetency of the femoral valves can be demonstrated. The technic of venogram to test deep vein function by injecting a foot vein, with a tourniquet at the ankle, is a rational procedure (see page 730).

The final decision as to whether or not the superficial femoral vein is to be ligated is made at the operating table. This is an important part of the operation. If the superficial femoral vein is essential to the patient for venous return from the leg, it should not be divided under any circumstances. In general this can be determined by the length of time that has elapsed since the acute process was active. Many serious complications have followed the ligation of the superficial femoral vein too soon after its occlusion.

Venogram in Operating Room.—With adequate and rapid x-ray facilities available in the operating room, the common femoral vein may be injected with an opaque solution. The patient is tilted so that the feet are down and the dye flows dependently by gravity into the superficial femoral or other branches. The proximal end of the vein can be occluded tempo-



Fig. 209.—Value of venogram before vein ligation. Incompetency of the femoral vein and its branches. Saphenous vein normal in size and valvular structures. Surgical treatment: elimination of incompetent femoral system. Note: If saphenous system had been eliminated, the pathology would have been compounded.

rarily. If the dye flows distally against the valves one can be certain that such valves are injured and incompetent.

Tests for Profunda Vein Adequacy.—A simple clinical test was evolved during the time such roentgen ray equipment was not available. Where its precepts are observed, it is a good test, and with its use at no time has a superficial femoral vein been ligated erroneously. In this test, a

silk ligature is placed around the superficial femoral vein just distal to its junction to the profunda. This ligature temporarily occludes the superficial femoral vein. As a rule, this superficial femoral vein then shrinks down in size. If the profunda vein is inadequate or the *superficial femoral vein is required* for the venous return, this latter vein will enlarge and begin to bulge immediately below the restraining suture. In such event, the superficial femoral vein should not be ligated. If it should be divided at such time, the leg will enlarge and there will be venous engorgement. It is suggested that no matter what other tests are used to determine whether or not to ligate the superficial femoral vein, this simple one be included.

OTHER TESTS FOR FEMORAL VEIN INADEQUACY.—(*a*) *Modified Perthes Test.*—This test has been described on page 564. Other tests are more accurate.

(*b*) *Femoral Venous Pressure.*[4]—It has been reported that venous pressure readings registered in the operating room will determine whether the operation should be done. If this pressure is over 30 mm. of water it has been described as indicating that the profunda system is inadequate and the superficial femoral vein should not be resected. We have had little success in interpreting this test on the individual patient, as often the *femoral vein is partly open, spastic, and no reading is obtained.* If an elevated venous pressure is present, the test is a sound one.

If the tests prove the femoral vein requires resection it is divided at the time the saphenous vein is resected. The saphenous system is resected at the femoral junction and at the incompetent points and stripped between these points.

**Superficial Femoral Vein Resection with Saphenous Vein Stripping.**—The combined superficial femoral and saphenous resection is performed by the following technic. A 3-inch longitudinal incision centering over the femoral saphenous junction is made in the groin. The superficial and deep femoral veins are exposed and examined. The superficial femoral vein is dissected free from the femoral artery which directly overlies it and this vein is ligated and transfixed with 000 silk. The vein is divided just distal to the profunda junction. The saphenous vein and its branches are divided at its junction with the femoral vein. All branches are widely resected as described on page 577 under the varicose vein operation.

Incompetent communicating vein points in the pathologic saphenous tree are likewise resected at the same time and the saphenous vein is stripped between the incompetent points as described on page 570 under the Treatment of Varicose Veins.

This is a rational procedure but should be performed only after examination shows an adequate femoral profunda vein to be present. After such an operation the venous drainage from the affected limb is effected by an adequate femoral profunda system and the efforts to recanalize this superficial femoral system then are ended. In fifty-five per cent of the patients with thrombotic ulcer so treated, healing of the ulcer has followed. Glasser reported 40 per cent healing.[4]

VENOGRAM IN OPERATING ROOM —With adequate and rapid x-ray facilities available in the operating room, the common femoral vein may be injected with an opaque solution The patient is tilted so that the feet are down and the dye flows *dependently* by gravity into the superficial femoral or other branches The proximal end of the vein can be occluded tempo-



FIG 209 —Value of venogram before vein ligation Incompetency of the femoral vein and its branches Saphenous vein normal in size and valvular structures Surgical treatment elimination of incompetent femoral system *Note* If saphenous system had been eliminated, the pathology would have been compounded

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**Ligation of the Popliteal Vein for Thrombotic Ulcer.**—The ligation and division of the popliteal vein as an adjunct in the therapy of the postthrombotic ulcer was advocated by Bauer.[1] This therapy was based on the fact that these ulcers occur in the lower part of the leg and that much of the back pressure can be relieved by division of the deep vein at this area. Others in this country have performed this operation and have reported on its success.[2] Our venograms on the patients who have had deep vein inflammation showed so consistently that the pathology involved the entire superficial vein that we divide the deep vein at the profunda level when such division is required at all. Division of the deep vein below the upper level of pathologic change appears inadequate and similar to dividing the saphenous vein at the knee. It took years to recognize the necessity of ligation at the saphenous-femoral junction. In selected instances, however, where the venogram shows the pathology is below the knee and the deep vein and its valves are functioning above the knee, the popliteal vein ligation and division is indicated.

**Sympathectomy for Postthrombotic Ulcers.**—In some cases of postthrombotic ulcer the outlined treatment will heal resistant ulcers. In others, where the thrombitis has been chronically recurrent and in those where there is a marked degree of spasm or where arterial failure is evident, sympathectomy must be performed to obtain and maintain healing of these thrombotic ulcers. This both improves the capillary circulation and relieves the causative spasm. Healing then may result promptly.

Interruption of the sympathetic system also is necessary in those cases of thrombotic ulcer where the skin is so wet and avascular that the ulcer cannot heal. These patients have a local hyperhidrosis.

**Skin Grafting.**—Where the ulcer has been present for a considerable time, there may be so much loss of skin that healing cannot occur and the epithelium itself must be replaced by skin grafts. This is similar to a loss of epithelium in burns. These grafts now are applied more often than before, since with the advent of the electric dermatome grafting is simple and over 97 per cent takes can be anticipated. (See chapter on Skin Grafting, page 741.) Skin graftings should not be done until the underlying pathology has been relieved as far as one is able to correct it and infection is no longer present.

**Excision of Ulcer and Grafting.**—If the ulcer has been present for a long time with many periods of healing and breaking down, the ulcer base becomes a hardened avascular scar. To heal such ulcers may be difficult or impossible even with skin grafts because this base is not a satisfactory recipient site. In such patients, the base of the ulcer and its edges are resected widely. When this area is cut away and the leather-like tissue removed, the underlying tissues will spread and herniate, showing how contracted they were by the old scar. Often feeding, incompetent veins are found which are basically pathologic. These can be resected.

The tissue left after such an excision will be found suitable for skin grafting. Skin grafts take even when such an excision exposes bone. Such grafts may be applied as exact fits, like bone grafts, using the electric dermatome. For details on the technic of skin grafting, see the chapter on Skin Grafting, page 741.

## FASCIECTOMY WITH LIGATION OF ALL THE INCOMPETENT COMMUNICATING VEINS

In addition to other ligations, Linton[8,A] has advocated the excision of the fascia and ligation of each incompetent communicating vein with a radicle exposure from the knee to the internal malleolus Where other therapy has been ineffective this treatment is of value We have used it with excision of ulcers without waiting for the healed epithelium which Linton felt essential and have found no untoward effects from this technic

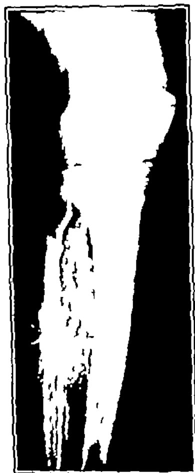


Fig 210a.—Incompetency of the deep vein system in the popliteal area. Note wide dilation and leak into the superficial system. Treatment resection popliteal vein and incompetent perforators with stripping of superficial system below the knee only

Thus the treatment of the postfemoral thrombotic ulcer is as follows (1) Resection of the superficial femoral vein if it was the original site of the pathology and remains so and if the femoral profunda vein is adequate (2) Resection and stripping of the saphenous system as previously described if this system is incompetent and venograms demonstrate an adequate deep vein system.

If healing does not occur then one or two or all three of the following steps may be necessary (1) sympathectomy (2) skin grafting, (3) excision of ulcer and grafting

FIG 210b —Venogram showing incompetent femoral system below knee In thigh, the saphenous is the only functioning vein Treatment resection of incompetency below knee, with retention of saphenous vein above knee



FIG. 211 —Typical phlebitic ulcers with secondary skin changes Treatment ligation of incompetent veins, excision of ulcer and skin graft

In many of these thrombotic ulcers only one of the above procedures will be sufficient to heal the ulcers In others however one must be prepared to continue the various operative treatments as described until the patient s ulcer area is healed and remains healed The surgical part is completed whenever such a result is obtained

## LIPECTOMY AND FASCIECTOMY WITH VEIN RESECTIONS

The disproportion in the size of the legs as the result of a thrombosis is annoying to many patients In addition lipomas often are present medially These may have been repeatedly bruised The veins under them are thrombotic and difficult to strip With careful measurements the excess tissue can be marked and excised including the skin fat veins and fascia The wound is plastically closed This procedure has been used routinely in the last 2 years. The scar in no way is as disfiguring or as disabling as the enlargement

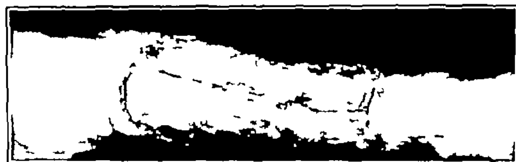


FIG 212 —Extensive ulceration treated by excision and electric dermatome graft after ligation and stripping of pathologic vein Graft attached in exact size Ninety nine per cent take

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# SECTION VI

## The Lymphatic Circulatory System

## Chapter 37

# SURGICAL TREATMENT OF LYMPHEDEMA (ELEPHANTIASIS LYMPH STASIS)

*Lymphedema Operation Applied to Other Lesions*
*Lymphedema Upper Extremity*

THE lymphatic system is an integral part of the circulatory system Its primary function is to return certain tissue fluids from the periphery to its corollary the venous system It arises like the vascular system by transformation of the mesoderm into endothelial areas which fuse into channels and plexuses It consists of capillary networks in the various organs and tissues collecting lymph vessels which carry the fluid from the capillaries eventually pouring the lymph into the venous blood stream Another one of the components is the lymph glands which filter the fluid as it passes through them and also supplies lymphocytes to the content of the lymph

Surgically we are interested in the lymphatic system because of

(a) its ability to localize or contain the spread of infection and malignancy and thus is a defensive aid

(b) its later transference as a pathway or even an accelerator system for the dissemination of infection and malignancy and

(c) lymph obstruction due to congenital or acquired diseases This obstruction may require surgical intervention

In vascular surgery our main concern is with the pathologic obstruction of the lymph Such obstruction is called lymphedema

**Definition.**—Lymphedema has been defined as a progressive histopathologic state characterized by a chronic inflammatory fibromatosis or hypertrophy of the hypodermal and dermal connective tissue '[19] Babcock[2] described it as a massive overgrowth involving the fibro-connective tissue of the skin subcutaneous tissue blood and lymph vessels with associated muscular degeneration and atrophy affecting the leg (elephant leg) scrotum (lymph scrotum) labia (lymph labia) The term elephantiasis apparently arose during the Roman army s campaign in Libya and this description came from their capture of natives who had these tremendous swellings of either the leg or scrotum in which not only the

size of the part but the contour of the skin reminded the Legion's observers of the hide of elephants, with which they had just come in contact

Lymph actually is the fluid which is contained in the lymph vessels This is separate from the plasma which is the fluid in the blood stream Outside of the lymph vessels and the blood vessels is tissue fluid An abnormal collection of tissue fluid from the blood vessels is an edema An abnormal collection of the lymph substances leaking out through the lymph vessels or their capillaries is called lymphedema When there is a large collection of fluid in the leg for any reason, often it is a combination of the increased tissue fluids from both the plasma and from the lymph vessels

To the surgeons, lymphedema may be considered as an obstruction or blockage in the lymphatic system which prevents the adequate absorption or return of the lymph fluid In this more specific instance we are concerned with the lymph collection in the extremities The lymph vessels in the skin are affected primarily Secondarily there is a marked increase in the venous components to aid lymph drainage, as this system acts as a corollary to the lymphatic system The lymphatic channels, after their invasion by inflammation of a primary or secondary nature, eventually heal by fibrosis and scarring The lumen thus is obliterated much as is the lumen after a thrombophlebitis in the veins Unlike veins after thrombophlebitis however, these lymph channels, in many instances, do not tend to recanalize, their entire structure being replaced by scar In fact, it has been shown in many of these patients that the tissue actually is deficient in lymphatic structures In others, the lymph vessels dilate distal to an obstruction This block may be a mechanical one due to scars, or it may follow the fibrosis of the lymph channels after a bacterial or parasitic invasion Massive venous thrombosis may cause secondary lymph obstruction as in so-called "milk-leg" Malignant infiltration of the lymph glands or channels may be the cause In others, the block cause is unknown, but by some predilection, the lymph obstruction occurs at about puberty, being thus familial or congenital, or both, in type (Milroy's Disease) Regardless of the cause, the final lymphedema picture depends on the site, degree and length of involvement of the lymphatics and the extent of the secondary recurrent inflammatory processes to which these patients become subject The part is enlarged tremendously and the skin pores are dilated and take on a pigskin- or elephant-like appearance The local circulation to the part becomes impaired, pigmentation is deposited with the edema, and frequently ulcerations follow When the extremity is raised the swelling recedes, the drainage taking place through the subcutaneous spaces to adjoining areas, *i e*, the abdomen, where there is no lymphatic obstruction On dependency the fluid re-accumulates Many of these patients become completely disabled We are not concerned in this book with the part the lymphatic system plays in draining the bowel through the cisterni chilæ to the thoracic duct

**Anatomy** —1 *The Lymphatic System of the Lower Extremity*—The lymphatic circulation is a part of Nature's defensive system It is composed of (1) lymph fluid, (2) lymph vessels, and (3) lymph glands The lymph fluid found in the closed lymphatic vessels is a clear, transparent,




FIG 213 —Anatomy of lymphatic system (Gray's Anatomy)

dorsum are joined on the forearm by those vessels draining the radial side of the forearm They then pass to the axilla Those from the third, fourth and fifth fingers run dorsally to the other side of the arm and up the flexor side to the axillary nodes Some of these chains run to the cubital lymph nodes Both the superficial and deep lymph systems of the upper extremity empty into the axillary nodes, although a few vessels may drain to the supraclavicular or the deltoideopectoral nodes At the elbow the deep and superficial nodes are connected [3a] After the division of such a chain the system early and actively attempts regeneration for seven to ten days

The *superficial* lymphatics form a dense plexus They unite to form trunks which accompany the cephalic, median and basilic veins A few from the ulnar side end in the supratrochlear glands, but most drain to the axilla The *deep* lymphatic vessels accompany the deep blood vessels and thus are the radial, ulnar, volar, and dorsal interosseous chains. There are few *lymph glands* in the superficial lymphatic system They are mainly the supratrochlear glands and a few deltoideopectoral glands Nearly all of the deep glands are in the axilla The anterior group is along the lower border of the pectoralis minor muscle, adjoining the thoracic artery They drain the anterior and lateral thoracic walls and the central and lateral parts of the breast The lateral glands are in relation to the axillary vein and drain the entire arm The posterior group of glands is close to the subscapular artery These drain the lower part of the back and neck The central glands are at the base of the axilla and drain from all the others There is a fifth group of glands posterior to the pectoralis minor These drain the upper and outer part of the breast

3 *The Lymph Vessels of the Genitalia* —The lymphatics of the genitalia are of surgical interest because of their importance in infection or the blockage due to parasites The lymph vessels of the perineum, the scrotum or vulva and the penis follow the course of the external pudendal vessels These glands drain into the superficial and deep inguinal glands The lymphatics from the glans penis and the clitoris drain to the deep inguinal nodes, as well as to the external iliac glands As is usual, the superficial lymph vessels follow the course of the superficial blood vessels and drain to the superficial inguinal glands The deep lymph vessels follow the principal blood vessels and drain to the subinguinal as well as to the aortic glands

**Physiology.**— *The Lymph Vessels* —The lymph system appears to admit substances into its closed system when objects are pressed against its surface The muscular contractions move the lymph, aided by the valves The lymph system may move rapidly or slowly depending upon the activity of the individual Lymph will coagulate when exposed to air but it takes from 2 to 4 times as long as blood, probably because there are no platelets present Infection and necrosis increase this coagulability The work of Homans, Drinker and Fields[12] showed that most lymphedema occurs as a result of thrombosis in the lymph vessel In their experimental reproduction of lymphedema, a valve in the lymph channels was destroyed by the inflammation they produced

## LYMPHEDEMA

**Etiology**.—We now believe that most lymphedema results from valve failure in the lymphatic system with obliteration of the lymphatic channels There are eight types of lymphedema

1 **Primary or Specific Lymphedema.**—Primary lymphedema is due to the specific invasion of the lymph vessels by the filarial worm *Wuchereria bancrofti* and at other times by the microorganisms *Mycobacterium tuberculosis* or the *Treponema pallidum* This lymph stasis was of military importance during World War II in the Pacific area In 1943 for example 5 646 battle trained Marines and sailors had to be evacuated from the South Pacific theater because they were affected with this lesion The contact part in the etiology of this picture was shown when after rigid quarantine from the natives only 743 were infected in 1944 [21]

When inflammation causes a valve failure the lymph pressure increases Lymph drainage is then attempted through lymph vessels which are not adequately supplied with valves There is an increase in the albumin and globulin content of the lymph and fibroblasts proliferate with a resultant fibrosis This thrombosis then reduces the resistance of the part and makes it more susceptible to attacks of acute inflammation The cause and effect therefore become cyclic It is quite certain that many reinfections are necessary to produce typical lymphedema This was demonstrated well by the fact that when these Marine patients were evacuated from filaria-infested areas the condition quickly subsided The filaria appears to be selective in its invasion of the lymph The scrotal area is particularly susceptible as are the lower extremities To produce the advanced picture of the diseased patient (typified by the patient sitting on his scrotum) he must be exposed for many years and have had repeated attacks of an inflammatory nature Often these filaria cannot be demonstrated *per se* but are encysted in Nature s effort to control the inflammation The condition appears to be epidemic in those who have been exposed The psychic effect of inflammation in the scrotum is well-demonstrated by these young men who feared most of all the loss of their virility A return of their morale after evacuation showed how much of a part this mental aberration played

The pathogenesis of the filaria by the mosquito is covered in texts on tropical medicine. The Treponema pallidum or the microbacterium tuberculosis may massively invade and block the lymphatic system but this occurs rarely

2 **Secondary or Infectious Lymphedema.**—Lymph stasis may be due to a secondary inflammatory invasion of the lymphatic system by non specific organisms such as the streptococci fungi or other organisms

The trichophytosis between the toes has resulted in attacks of acute lymphangitis Such growth may merely break the skin and permit the entrance of other more active infecting organisms At times an inflammation can be minimal and appear to attack only the lymph vessels themselves A slight injury to bone for example may expose the lymphatics to inflammation Fibrosis and scarring occur secondarily

3 **Traumatic Lymphedema.**—Injury with scarring following injuries, operations, keloids, burns, and excess exposure to roentgen rays, radium or radioactive isotopes may cause lymph collections

Excision of lymph nodes for infection, cancer, tuberculosis, or other disease is a common cause for lymph stasis The rarity with which such lymph stasis occurs however is further evidence of the ability of the body to develop collateral lymph channels in most instances The burn from x-ray or radium therapy particularly causes such lymphedema by sealing off the lymphatic channels, which is one of the therapeutic functions of



Fig 215 —Congenital Lymph Stasis Boy, age ten, born with cystic hygroma—collections back of neck, enormous lymph stasis on left arm and hand (Courtesy of Dr Bruce Martin of Columbus, Ohio, and Dr John Conley, New York City )

such therapy in cancer Lymphedema was common after radioactive exposure in the atom-bombed cities of Hiroshima and Nagasaki It is noteworthy that keloid formers are much more subject to lymphedema, the keloid acting similarly to the radium or x-ray in sealing off the lymphatic channels Poor coaptation of scars and inadequate approximation of tissues is a cause

4 **Congenital Lymph Collections.**—These accumulations may be the result of cystic hygroma, and possibly Milroy's disease fits into this category Many abnormal collections of lymph have been described in other parts of the body. Some of these are amenable to excision and others, because of their involvement of important structures, are not removable.

5 **Allergic Lymphedema.**—In the allergic patient exposure to cold to drugs environment change or to other precipitants to which the patient may be sensitive may cause the picture The allergic factor should be considered Lymphedema of the arm frequently may be caused by an allergic manifestation At times a therapeutic test with one of the anti histamine drugs will help in the differentiation

6 **Malignant Lymphedema** —Infiltration of the lymphatic system by malignant growth will cause lymphedema Surgeons are aware of the lymphedema occurring in the arm after a radical breast resection for carcinoma Most of these blocks are due to recurrent malignancies and the burden of proof in such instances *is that they are not due to metastatic* node involvement Years ago Halsted[8] studied the lymphedemas fol lowing breast amputation and decided they were due to infection by strepto-cocci He thought that this was due to the fact that the glands in the

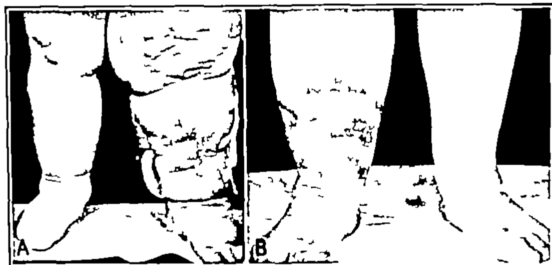



Fig 216 —*A* Enormous lymphedema after a thrombitis secondary to trauma *B* Postthrombitic lymphedema

axilla had been removed and therefore the defensive factors were no longer present Most of the cases he reported in this category later died of recurrent malignancy The tremendous lymphedemas of the lower extremity and vulva after carcinoma of the pelvis are familiar pictures these days in which all types of radical surgical and radiological therapy keep patients alive for longer periods of time A single negative biopsy is not sufficient to rule out metastatic involvement as a cause for lymph edema following a carcinoma Sarcoma also is a cause for lymphedema The malignant lymphomata will cause lymph obstructions (lymph sar coma Hodgkin s disease)

7 **Postthrombotic Lymphedema** —There may be lymph collections following massive clotting of veins The lymphedema which follows venous obstruction is a familiar complication It is part of the picture of milk leg With the vein blockage more drainage by the lymph system is re-quired, which in turn backs up or fails The differentiation of this swelling

and the management of this condition have been extensively studied lately The tissue fluids accumulate due to the vein block Lymph drainage becomes inadequate [28,33]

8 **Idiopathic or Essential Lymphedema.**—Some lymphedemas are of unknown origin and Milroy's disease is in such a class [20a] Milroy originally described the idiopathic or essential lymphedema as one in which both a familial and a congenital history was present Allen, Barker and Hines[1] differentiated congenital lymphedema into simple and hereditary types The term "simple" was reserved for those in which only one member of a family was so affected and "hereditary" for the group described originally by Milroy in which many of the same family were involved In the pure congenital type, the condition is present at birth Lymphedema is char-

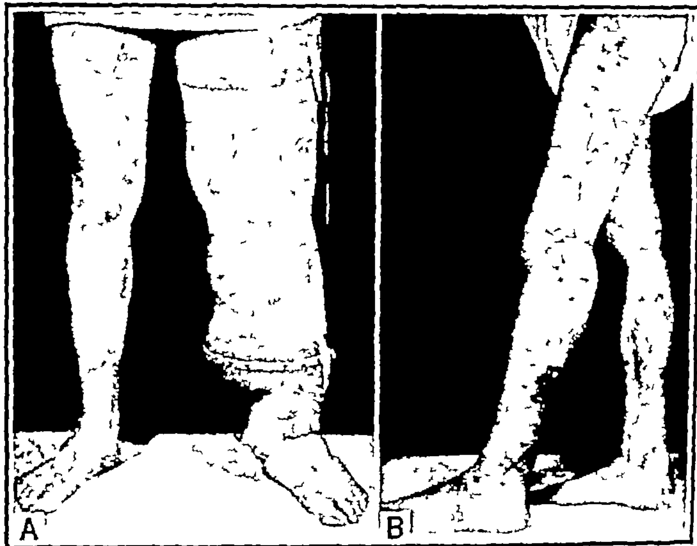



FIG 217 —*A*, Idiopathic lymphedema One leg was 38 inches in diameter, the other 13 inches *B*, Operative removal of the lymphatic collection (Photograph shown by courtesy of Dr A Wilbur Duryee, in Pratt, Surg, Gynec and Obst )

acterized by swelling of the part and often by repeated attacks of acute inflammation In the simple congenital type, the skin and the deep fascia are separated more widely than usual and the ordinary fat tissue is replaced by large lymph channels The lymphedema appearing at puberty has also been considered by many authors to be of the Milroy type In these instances there may be a mild lymphedema present at the time of birth, but this is not too apparent to the patients until such time as the normal changes of puberty emphasize the disparity in the size of the limbs We consider these two types of lymphedema as one It is an interesting but still little understood lesion Why so many of these patients do well until puberty, before the obstruction appears, is not clear This points to an endocrine dysfunction as a cause Some congenital defect in the anatomic and physiologic mechanism of these patients is shown by the familial tendency.

**Pathology** —The pathology of certain types of lesions, such as those following x ray or radiation or carcinomatous invasion is obvious The pathology which occurs after a primary or secondary infectious invasion also is well known

1 **Pathology of Infectious Lymphedema** (Primary or Secondary Infections) —The organism either by direct invasion when there is an open lesion or by being absorbed through the cellular lining in the lymphatic vessel inflames the lymph vessel The organism multiplies and there is a reaction



Fig 218 —Lymphedema in child present at birth. (Courtesy of Dr J M Baker Springfield Mass.)

of the lymph fluid and the lymph vessel to its presence The number of cellular elements increases there is edema, and swelling and extension of the process proximally The degree of inflammation varies with the type and virulence of the organism and the defense of the host The inflammation may spread to the parent glands and stop there. It may spread further and be accompanied by a perilymphangitis and lymphadenitis With this inflammation there is a lymph blockage and an increase in the tissue fluid as the lymph component is not carried away and lymph leaks from the vessels The edema is a hard brawny type which pits with difficulty In extensive cases there may be suppuration A relative venous obstruction



FIG 219 —Tremendous congenital lymphedema of fingers, hand and forearm (Courtesy of Dr Bruce Martin of Columbus, Ohio )

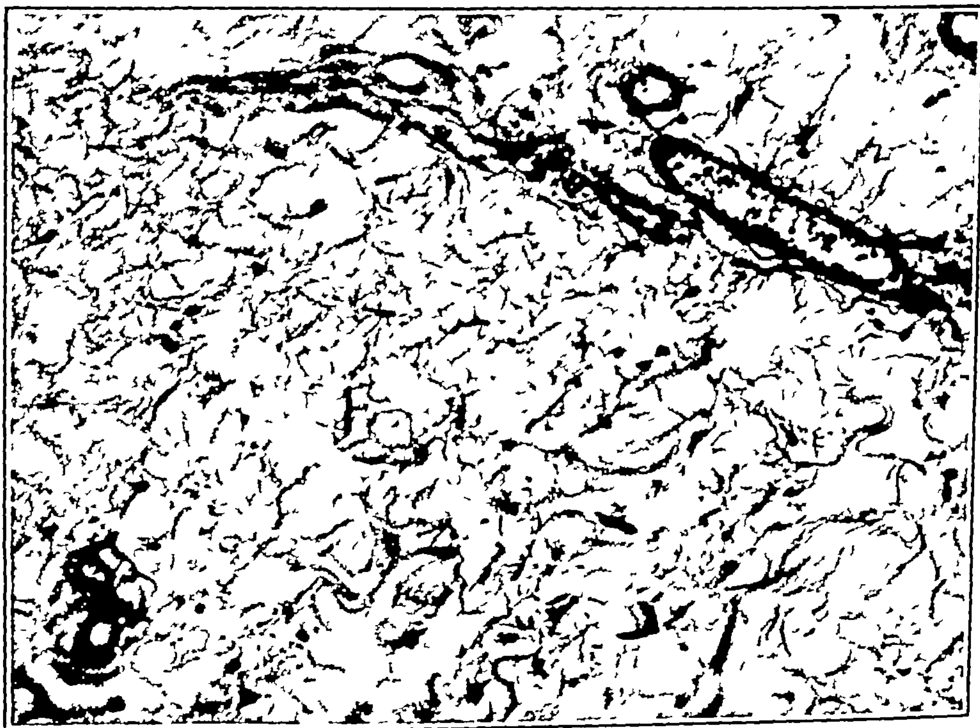


FIG 220 —Pathology of lymphedema condensation in avascular subepidermis layer

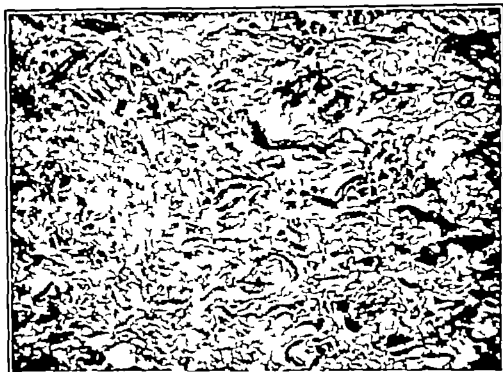


FIG 221 —Pathology of lymphedema fragmentation of collagen due to pressure of the lymph.



FIG 222 —Pathology of lymphedema vascular infiltration in the dermis layer

results from the pressure of the lymph stasis and an overstrain on the venous return The veins then dilate and form new collaterals The fate of the lymphatic vessel after the infection or inflammation depends upon the degree of irritation and its previous normal or damaged status With healing, there is a scarring and fibrosis The lymph valves are involved in such a process, and the valves may be permanently damaged or destroyed The lymph vessel itself may be replaced by fibrosis and scar If sufficient numbers of lymph vessels are so affected, the lymph drainage through such conduits cannot be re-established The dermis, fat and subcutaneous tissues are replaced by tissue spaces in which fluid accumulates Fibrous trabeculæ grow into these spaces and form bands Later these serve to keep the tissue spaces open, and in the final stage the lower parts of the skin are held away from the fascia by well-formed and scarred areas which fill with fluid upon dependency and to some extent empty with elevation During this elevation, the fluid will flow gravitationally to areas in which the lymphatic drainage is unaffected There may be secondary effects on the muscles, tendons, bones and joints due to pressure, atrophy and disuse The nutrition of the skin is affected Its pores dilate (pigskin) and there may be ulceration Lymph may run from such areas in large quantities

2 **Pathology of Postthrombotic Lymphedema.**—The changes accompanying the postthrombotic syndrome have been studied by many clinics, including our own [30,31,33] There is a wide replacement of the dermis and subcutaneous tissues by a combination of lymphatic and venous fluid Between the epidermis and the deep fascia there is a marked fibrosis, the fibrous changes being so extensive as to lift the skin from the deep fascia In these spaces there is a combination of fluid both of the normal edema and lymphedema type There is extensive effort at collateral development both on the venous and lymphatic sides The skin changes are of two types There are those associated with a marked venous congestion in which there are pigment deposits and the skin has a brawny induration and is subject to ulcerations, and there is a second type in which the pathologic picture is that of a large, white, cold leg, extremely sensitive, in which the swelling completely covers the engorged venous elements Spasm of both the arteries and veins is a typical part of this syndrome Causalgic-like response is made to any stimulus Many of the veins in such a picture are partly recanalized and partly thrombosed There are liquid and clotted elements and some of the clots in the veins have been replaced by fibrosis There may be a lymph coagulation or there may be multiple collateral lymphatic channels, and the collections are then due to faulty lymph valve structures in these collaterals The skin and subcutaneous tissues are thickened markedly Muscle may atrophy and the nerve fibers and sweat glands likewise may be destroyed by the pressure of the fluid accumulations There is marked infiltration of lymphocytes and at times leukocytes The pathologic picture varies if there has been a recent inflammatory process The signs of an acute inflammation with edema, marked round-cell infiltration and leukocyte penetration, diapedesis of cells through the walls and secondary inflammation in the lymph channels then appear

3 **Pathology of Milroy's Disease** —In the pathology of Milroy's disease the pathologic section (Fig 220) shows wide epidermic and subepidermic layers There is great thickening with obliteration of the papillæ and absence of the sweat glands Under this layer is a marked condensation and avascularity, with the blood vessels being obliterated by pressure of the edema In a second section (Fig 221) the traumatic effects of the edema are shown with fragmentation of collagen Figure 222 shows microscopically the vascular infiltration in the dermis layer which occurs with lymphedema In another photomicrograph (Fig 223) the enormous dilated lymph vessel with the normal components of its walls missing and vacuolization is demonstrated The fat first may be pressed to one side and then replaced by the lymph vacules Figure 224 shows the end result in which fibrous trabeculæ have formed in the subcutaneous fat These fibrous bands hold the tissue spaces open and fluid collects in these spaces when the part is dependent Sometimes the fat sloughs In others the muscles and tendons and bones will show the atrophy and also other evidences of pressure and disuse as the lymphatic blocks have developed The inguinal and other lymph glands are enlarged and later become fibrotic. The lymph channels may be entirely replaced by fibrosis or become secondarily closed after the inflammation has occurred repeatedly

**Treatment.**—Whenever possible the obvious treatment is to correct the underlying cause. In the specific or primary lymphedema caused by filaria the contact with those infected with filaria for example must be broken Syphilis and tuberculosis require specific therapy Allergists may be of aid in the sensitization group Antihistamine drugs may help Exposure to increasing cold may desensitize this group Antibiotic therapy is of great help to relieve the repeated bouts of sepsis which occur as the lymphatic defense decreases. Inflammation increases the pathologic changes This therapy should be continued in some cases for years Plastic excision and correction of obstructions burns or scars causing lymph stasis may be necessary In addition to these fundamental measures once the lymph obstruction has developed and the lymph conduits are closed and after the possibilities of conservative drainage have been exhausted other aids in the correction of the end status of lymphedema exist.

**Conservative Therapy** —(*a*) *Elevation and Support* —If the lymphedema is mild as it is in many cases simple medical measures of support frequently will take care of the problem The fundamental part of this treatment is to elevate the part sufficiently to drain the area and reduce the swelling and adequately support the part when the extremity is in a dependent position These two points need emphasis *Elevation of the extremity* means that the part should be raised *above heart level* always at night, and each time swelling occurs during the day At first this may require ten or twelve periods of elevation a day The lymph fluid cannot be permitted to remain in the subcutaneous tissues for any length of time, because if it does fibrosis will occur and a permanent subcutaneous tissue space will form in which fluid can collect. The term *adequate support* needs clarification The support must be sufficient to maintain the decompression once the elevation has eliminated the fluid If swelling occurs (even with the adequate

support), as shown by symptoms of fullness, tightness, pressure or pain, the part must be elevated again above the heart level until the symptoms caused by that swelling subside An Ace-type bandage or rubber bandage is used and is preferred to a fitted elastic stocking at this stage, because the elastic or rubber bandage can be adjusted during the day to take up any slack after the swelling has gone down upon elevation. With a fitted elastic stocking the swelling will always occur to fill the size of the stocking If the space between the skin and the subcutaneous tissues is obliterated *early* in the disease in this way for a *sufficient length of time*, scarring may occur and the space be closed permanently In most of the patients with the postthrombotic syndrome of so-called "milk leg," a program such as this, if instituted early and followed through long enough, will be effective

(*b*) *Vasodilatation* —The local and general vasodilators have not been too effective in the therapy. If spasm alone is a factor, or where the cause is mainly venous, they may be of some help Mecholyl acetyl-beta-methyl chlorine given by iontophoresis has helped some of the mild group The sympatholytic and adrenolytic drugs have been of little if any help Warm packs, soaks and warm baths may aid

(*c*) *Massage* —Mild massage from the distal to the proximal part will be of assistance if such massage is not traumatizing This massage must be light, regular, and done by an interested, careful person Walking in a lake or ocean in water up to the waist will help, as the pressure of the displaced water helps press or massage the fluid out of the tissues

(*d*) *Antibiotics* —Due to the lymphatic blockage, the defensive part of the system is curtailed As a result, severe inflammation, periodically, may occur in the leg These irregular septic bouts require repeated courses of a specific antibiotic therapy with the selection of the drug best suited to control the organism in each patient By an analysis of the frequency of the attacks, the drug can be administered prophylactically just before the fever bout should occur Often, when the attacks are prevented for a time, they do not recur This sepsis occurs as cellulitis or thrombitis (thrombophlebitis) and often is diagnosed as "erysipelas" The temperature may reach as high as 106° After each attack there will be more residual and permanent lymphedema due to the new blocks caused by the inflammation

(*e*) *Sympathetic Nerve Blocks* —In those patients who have a lymphedema from the postthrombotic syndrome, warm packs and repeated lumbar sympathetic nerve blocks may help by relieving spasm and dilating the collateral vessels

**Surgical Treatment** —(*a*) *Surgical Sympathectomy* —The patient with a moderate type of chronic lymphedema and those with swelling after the postthrombotic syndrome may respond well to surgical sympathectomy This measure has been discussed elsewhere (see pages 674 to 675). In the mild lymphedema patient where the pathology is progressing, a sympathetic block may reduce or relieve the pathology markedly This therapy is suggested also for those patients in the younger age group and should always be tried before more radical excisions and grafting procedures are

undertaken In our series of patients 22 have been in the age group between ten and twenty Of these young individuals over one-third have responded so well to sympathectomy and support that their problem no longer is a serious one Many of these patients particularly the girls are at the age when appearance is most important If sympathectomy and support controls the problem such therapy should be considered conservative The mechanism by which sympathectomy aids lymph drainage is not clear It is likely that the increased vascularity on both the arterial and venous sides aids in absorbing the lymph accumulations and that more of the tissue fluids flow more rapidly through the venous channels than prior to sympathectomy Spasm is also relaxed [23,30,31,33]

**Surgical Excision.**—The patients with massive lymphedema who are physically and psychologically disabled by the gross size of the legs require surgical correction of the residual lymph mass for physical comfort and rehabilitation Surgical literature is replete with the efforts of the early surgeons to control this condition Lisfranc's[17] attempts at scarification Carnochan's[4] ligation of the external iliac and femoral arteries and the other somewhat bizarre attempts showed the intense desire of the patient for relief and of the surgeon to supply it Handley[9] was the first to try to drain the blocked lymph to areas where there was no lymphatic obstruction his drains being subcutaneously-placed silk threads More recently this method has been revised using steel wires and needles without satisfactory results. Lexer[16] Walther[33] Lanz[14] Rosenow[24] and Oppel[22] are all familiar names in the attempts at surgical aid Lexer[16] suggested the possibility of removing the deep fascia and Kondoleon[14] whose name is associated with the classical operation incised a small window in the deep fascia and removed a section of the edematous subcutaneous tissue and fat, then replaced the skin on the muscle He reported only 6 such operations Sistrunk[27] Homans[10,11] Ghormley[8] Matas[19] and Macey[18] have all supplied valuable contributions to the surgical correction of this problem

Our interest in this problem began in 1936 with the addition of surgical components to a large Vascular Clinic In this Vascular Clinic were found many hapless, and somewhat hopeless individuals with lymphedema of the lower extremities Their previous therapy had been that of support. Their affliction was so great that they were willing to try any type of procedure if it offered hope of relief Our first report in 1939[28] dealt with 12 modified Kondoleon operations and the following year[27] in conjunction with Irving Wright, 8 more were added to this list No operations of this type were performed from 1942 to 1945 due to Navy service but by 1949 26 more procedures of one kind or another for relief of lymphedema had been performed. It became obvious that the surgical principle in the treatment of lymphedema is twofold The voluminous skin subcutaneous and lymph tissue must be excised Efforts must be directed to obtain drainage for the epithelium of the skin through other lymphatic channels The lymph channels particularly in the muscle offer such drainage possibilities inasmuch as the lymph pathology is in the superficial lymph channels above the deep muscles but particularly in the skin Lymph obstruction does not appear to occur below the subcutaneous level nor does it follow the perforator lymph branches into the deep lymph channels Several surgical

procedures have been described and they will be reviewed briefly In addition to surgical sympathectomy, which has been performed 165 times for the swelling of the postthrombotic syndrome, the following measures have been utilized (See pages 674 to 675 )

1 *Total Fascia Excision Procedure* —This procedure was discussed in 1940[26] and 1941[27] by the author It was an extension and combination of other modified Kondoleon[14] principles and particularly that of the Homan's operation [10,11] In this operation, a sufficient ellipse of skin was removed to overcorrect the leg after its excision The skin was raised as uniformly thin as possible from the subcutaneous tissue in the thigh until at least 80 per cent of the thigh had been encircled, and in the calf the entire circum-



FIG 223 —Pathology of lymphedema dilated lymphatic vessels and spaces filled with edema

ference of the leg was so treated The incision was then continued down through the subcutaneous tissue, superficial fascia, lymph, fat and deep fascia and this entire area was removed *en bloc* This left only the full layer of skin and the denuded muscles The skin was then approximated over the bared muscles with interrupted steel wire and was held against the muscle by pressure bandages In this way it was likened to an immense *full*-thickness skin graft This procedure was used in 38 instances and the result was improvement in approximately 60 per cent of the patients. The disadvantages of this operation were apparent at once To completely denude the skin is impossible The thickness of the skin necessarily varied Technically, it was a difficult procedure Full-thickness grafts do not take well, and the leg configuration later was not all one would wish In addition, there was some recurrence

Our pathologic sections showed that the lymphedema began in the dermis approximately 1/16 of an inch below the surface. Thus in our undermining operations we were leaving pathologic lymph tissue in the skin and then replacing such tissue on the muscle. The lymphedema therefore was left on the patient and only the extensions of it to the subcutaneous and fatty tissues were excised.

2. *Free and Pedicle Grafting Procedures.*—It was evident very early in this work that some of the undermined skin would slough and free grafts and/or pedicle grafts would be necessary to supply the lost skin. The pedicle of the graft was placed above the blocked lymphatic area and the skin seemed to heal much better and there was less residual edema. The lymph drainage was obtained by the skin continuity to unblocked lym-

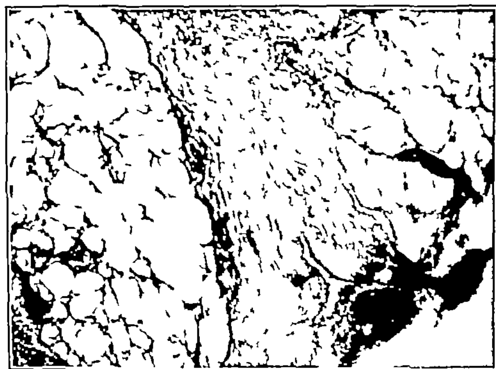


FIG. 224.—Pathology of lymphedema: thickened fibrous trabeculæ in subcutaneous fat. These fibrous bands are end results and tissue spaces remain between them for collection of fluid when the part is dependent.

phatic channels. This surgical procedure was difficult, had to be done in stages and was time-consuming but was frequently the only procedure which could be utilized.

3. *Dermatome Grafts.*—In 1948 Macey[14] called attention to a variation in the technic in which the skin, after the subcutaneous and deep fascia were excised, was replaced by Paget dermatome graft directly on the muscle. This operation had its limitations as Paget grafts often could not be raised from the lobular tissues usual in lymphedema. Often much of the rest of the body had to be used as donor sites. This method removed the skin and subcutaneous tissues. It was applied by Bunnell[14] to the upper extremities and by Poth[24] and later by Blocker[11] to the lower extremities. This method did not work well in our experience because the deep fascia

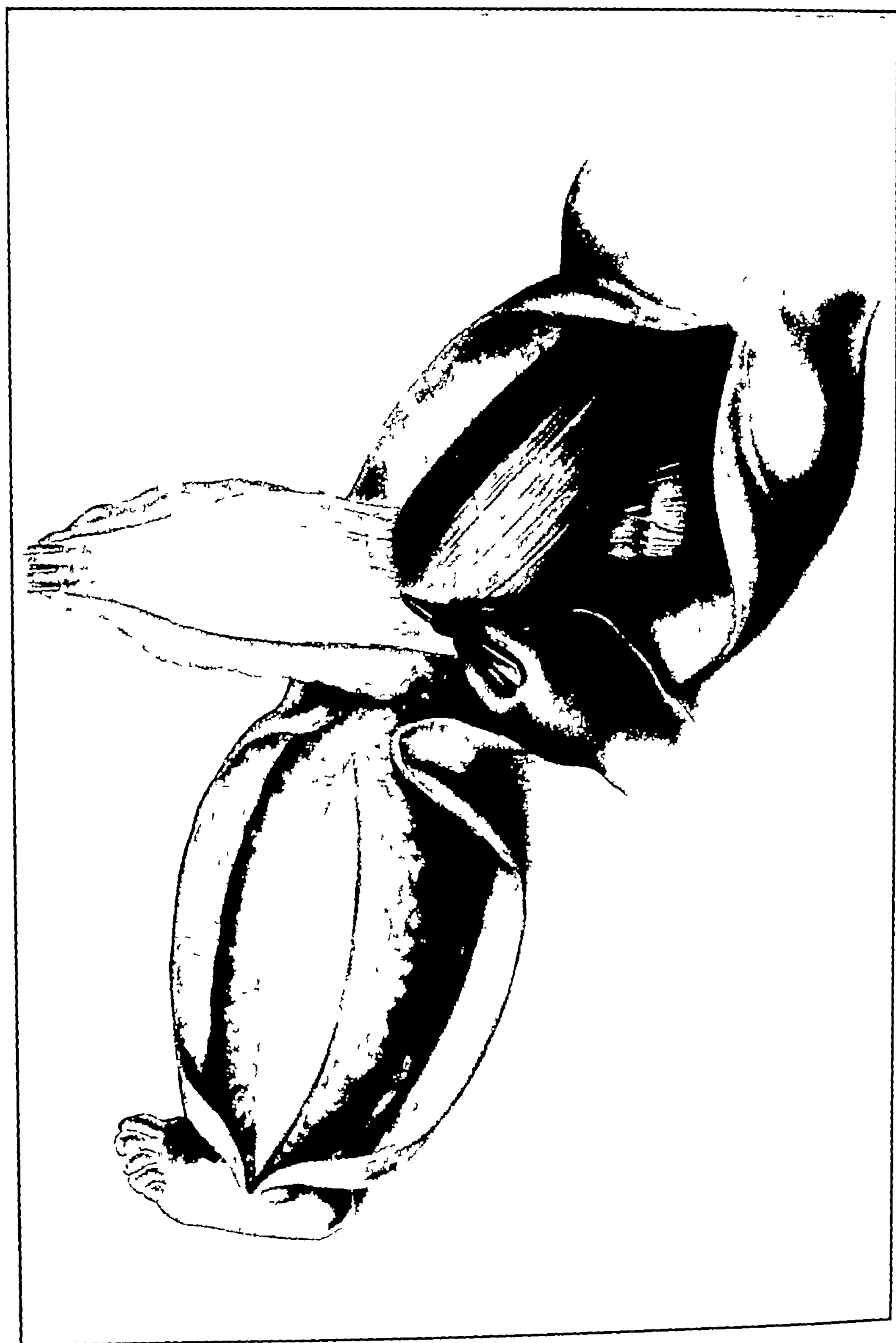


FIG 225 —Old method of treatment Full-thickness skin layer was raised Superficial and deep fascia thereunder excised approximating skin onto muscle Operation failed because pathologic lymphatic components of skin were not removed.

was not removed, and the skin grafts, as raised, often were not sufficiently thin to exclude the lymph in the skin

4 *Pratt Lymphedema Operation (Electric Dermatome Method)* —It was apparent from long follow-ups that the operations we had utilized so far for lymphedema were incompletely successful Biopsy specimens showed that the blocked lymphatic structures in the skin itself had not been removed [23] (See Figure 227 ) As a result with dependency the same lymph stasis existed Our object was to remove just the epithelial part of the skin and then excise all the lymphatic portion of the skin with the subcutaneous and superficial and deep fascia and fat tissue Our repeated

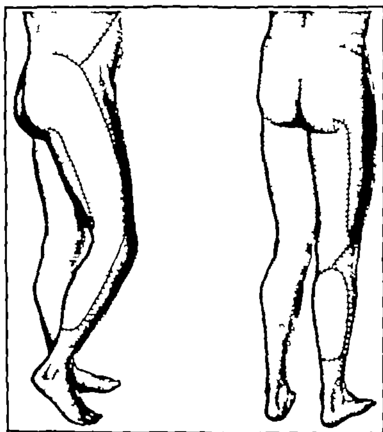


FIG 226 —Old method. Large pedicle grafts with base above lymph block Therapy technically difficult and frequently ineffectual

biopsies showed that the lymphedema accumulation did not occur in the epidermis They began in the dermis at approximately $\frac{1}{16}$ of an inch below the surface It was necessary therefore that the epithelium which was going to be replaced on the muscle should be shaved thin enough so that it would include no pathologic lymph collection The electric skin dermatome permitted such uniform excisions

In 1949 the electric skin graft instrument of Brown appeared [24] With this instrument, long uniformly thin skin grafts could be raised quickly the length of these grafts varying only with the availability of tissue from which the skin is to be removed This type of electric dermatome has

revolutionized plastic surgery Grafts can be made to fit the recipient site with a cabinetmaker's precision The treatment of lymphedema and associated massive tissue collections (hemangiomata or congenital arteriovenous fistulas) now can be attacked with more certainty of success These dermatomes will take skin from curves, over joints, and bone prominences and in and out of concave or convex surfaces In lymphedema, the pores are very wide This does not interfere with the taking or healing of the grafts We now operate these legs in two stages the first to include the anterior upper and lower leg, the second the posterior parts This change makes grafting more simple

In some cases the grafts are tacked lightly and left open

**Technical Points.**—(1) *Anesthesia* —Continuous spinal anesthesia is advocated as the ideal anesthesia This is given by a catheter inserted through a needle in the dura, the needle then being withdrawn Fractional doses of anesthesia can be given during the long time the patient is on the table This anesthesia minimizes the shock element of the operation To this is added hypotensive anesthesia in those whose general status permits it (See chapter on Anesthesia, page 27 )

(2) *Blood Transfusion* —This quantitative replacement of blood needs great emphasis, since only by weighing the sponges and clots can the amount of blood loss be realized The large areas of denuded tissue attendant to the operation and the loss of lymph and serum make blood loss more important All sponges, clots, towels, etc , should be weighed from time to time, suction bottles checked and the blood replaced quantitatively pint for pint regardless of the patient's color, pulse or blood pressure The apparently innocuous and continuous loss of blood and serum may not be a registerable factor for some time, and then a sudden profound shock may occur, which at times may be irreversible The blood replacement factor must be fully appreciated and carefully controlled because the blood and fluid loss in these patients is enormous and often not noticed The plasma expanders may be used if blood is not available (See pages 43 to 44 )

(3) *Skin* —The extremity should be elevated for approximately two weeks to permit the lymphedema to drain Skin preparations during this time should be performed with a sulfonated detergent (pHisoHex), soap and water, and also with potassium permanganate soaks (1 20,000) to restrict fungus infections and to dry up areas made moist by the infolding of the heavy skin Antibiotics should be given prophylactically in large doses Cultures should be taken of any ulcerations and these should be made surgically clean by the soaks and by the local application of antibiotics If there are ulcers or pruritis, muscle adenylic acid (introduced in the Vascular Clinic of St Vincent's Hospital in 1950)[32] has helped to heal the ulcers This substance reduces itching and weeping and ulcers close more rapidly While the mechanism of its action is not clear, it may act by supplying some cellular factor absent in ulcers of this type Just before operation, the skin should be washed for three minutes with a sulfonated detergent (pHisoHex with Hexachlorophene, 3 per cent)—all other skin preparations or paintings have been discarded

(4) *Position of Patient* —It is technically difficult to correctly position these patients for the removal of the grafts and tissues and to replace the grafts. We have found that the use of an orthopedic table works best. The leg is thoroughly prepared a glove applied to the toes and the entire extremity placed through the opening in a laparotomy sheet The foot is elevated so that one has access to all sides of it and the leg Elevating the part during the operation reduces some of the blood loss

(5) *Surgical Technic* — The skin is removed in strips 4 inches wide by $\frac{12}{1000}$ to $\frac{18}{1000}$ of an inch thick A new blade should be used in the dermatome each time The surgeon himself should assemble and set the derma-



FIG 227 —Biopsy specimen showing lymph collection in the skin Section taken at junction of operatively-corrected and non-corrected lymphedema Area to the right has had skin subcutaneous and deep fascia removed and has been grafted Area to the left has not been operated.

tome and the moving part should be lubricated with mineral oil. Grafts lift best when sterile mineral oil is applied to the skin As the skin comes off it may be held up by mosquito hemostats. The skin should be covered with saline solution The rest of the skin subcutaneous tissue fat lymph and fascia are then excised en masse Since a tourniquet cannot be used one must be prepared for extensive bleeding *Skilled assistants* and a sufficient number of *hemostats* are mandatory We have used over 400 hemostats at one time In the thigh a tension on the tissue being excised creates a line of cleavage In the calf however some of the deep fascia also surrounds the muscle bundles and the dissection is more difficult. Care must be taken near the *peroneal nerve* where it winds around the neck of the fibula. The

*ankle* and *knee joints* create special problems The foot is treated the same as the leg Any residual swelling in the toes can be controlled later with a leather shoe for support Silk or cotton sutures are avoided as they may create sinuses. The ties should be of fine catgut (000) The skin is best sutured with fine silk or steel wire (# 37), which has minimal tissue reaction. A pattern of the denuded muscle is cut Strips of skin are sutured together to fit the pattern

We have found it easier technically to operate on these legs in two stages, performing the anterior half of both the thigh, calf and foot, and secondarily operating on the posterior half



FIG 228 —Congenital lymphedema of left arm Indian girl, age four Dermatome removal of skin and excision of rest of skin and subcutaneous tissue *without* removal of deep fascia resulted in recurrence (Courtesy of Dr Alexander Bill of Seattle, Wash )

(6) *Adequate Support* —Adequate support is used after the operation We have not had lymphedema re-accumulation at these areas The patients have been observed now four years without any signs of recurrence if the technic was followed correctly The only complications have been some keloid formation at the site of the sutures and some dermatitis Our present problem is trying to avoid such keloids by putting these grafts on without sewing This is important and its principles must be understood (see page 744)

**Complications of the Operation** —(1) *Blood Loss* —Ample blood replacement is required Skilled assistants are necessary to reduce the blood loss Further reduction in the blood loss is achieved by hypotensive anesthesia This is made possible by one of the drugs discussed under hypotensive anesthesia, by spinal anesthesia, or by refrigeration.

(2) *Thromboembolism* —Because of the large number of vessels to be clamped and ligated and the immobility after operation this problem must be kept in mind Movement in bed is encouraged and antithrombotic drugs to a low therapeutic level are employed

(3) *Shock* —This complication always is possible because of the extensive denuding of large portions of the body the blood loss and the length of time the operation requires Hypotensive anesthesia is another factor Attention to the blood loss is a primary requisite We feel that spinal anesthesia is an important measure in preventing shock The two-team operation and sufficient assistance shortens the procedure and counteracts the possibilities of shock

(4) *Graft Loss* —Very few grafts will be lost if the technic is followed carefully We save all extra skin to replace any graft which may be lost We have found it wise to graft the foot with skin taken from the foot The texture of the skin over the foot is stronger and more able to stand the irritations of wearing a shoe and walking

(5) *Keloid Formation* —Keloids occur only in some patients It is likely that they are a complication of some inadequacy in the skin texture They appear more often however in lymphedema operations than they do in general surgery Most of them are at the site of suture and will be eliminated once we are able to apply these grafts without the extensive sewing now required

(6) *Residual Scars* —These scars can be covered cosmetically by Cover Mark and other creams and powders A well fitted elastic or nylon stocking also helps Keloids sometimes must be excised and then treated with x ray Electrically-driven pumice will eradicate some keloid scars

CASE REPORT —The treatment of a male age forty-seven is given in detail as an example of the possibilities of this new operation The circumferential measurements of this leg taken at the six lobular areas on the anterior surface were as follows 106 cm 90.6 cm 104 cm 101 cm 83.5 cm and 76.5 cm in comparison with the left leg which measured 71.4 cm 61.5 cm 52.5 cm 40.2 cm 38.6 cm and 24.8 cm This lymphedema had been present for thirty years Ten years before we were consulted some type of a modified Kondoleon procedure had been performed without benefit to the patient The irregularity of the skin the incisions made at the previous plastic operation and the monstrous lobular appearance made any of the operations previously described technically impossible In addition the previous procedure had resulted in an infection and as a result the line of subcutaneous and fascial plane cleavage was no longer present This patient was operated in three stages as follows

*Operation Technic* —*In the first stage* 250 square inches of skin were lifted off the calf with the electric dermatome The sections of grafts varied from 10 to 15 inches in length and were all 3 inches wide and $\frac{18}{1000}$ of an inch thick. The grafts were sutured together with fine silk All of the rest of the skin the superficial and deep fascia including the lymph deposits from the knee to the ankle then were surgically excised The leg was reduced from a circumference of 33¼ inches to a circumference of 17 inches which compared favorably with the opposite calf circumference of 16 inches. The skin was then replaced directly on the muscle sutured in place

with fine silk in a continuous fashion All 255 square inches of skin were used. Pressure dressings were applied, antibiotic therapy was continued and the wounds and the grafts were not disturbed for a period of fourteen days At the end of that time, it was found that approximately 97 per cent of the grafts had taken and the small areas which did not take were mostly at the site of sutures.

The second stage was performed three weeks after the first procedure, 198 square inches of skin was lifted from the anterior and posterior medial

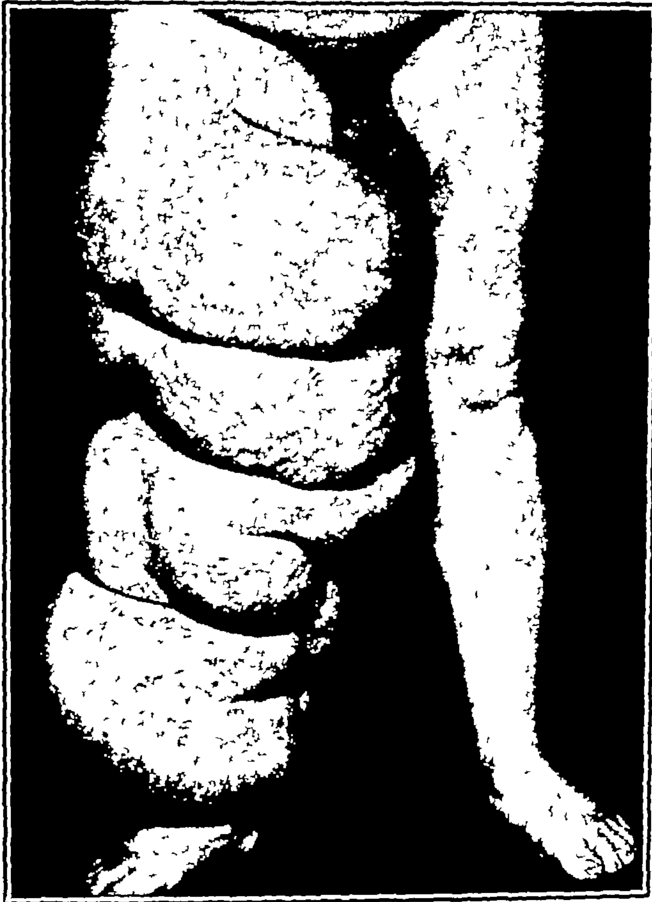


FIG 229 —Massive lymphedema operated in 3 stages 110 pounds of tissue removed (Pratt, courtesy of J A M A)

surface of the thigh The underlying tissue removed weighed approximately 25 pounds, with an equal weight being lost in fluid The grafts near the knee were placed transversely There was approximately 95 per cent graft take, only one graft being partly lost. An attempt was made to take two grafts $\frac{17}{1000}$ inches deep from the same donor site in this operation, and it was one of these second grafts which did not take well

At the third stage, 194 square inches of skin graft were raised and replaced on the denuded muscle tissue Several pieces of skin saved from the second stage operation which had been kept under sterile precautions in the refrigerator were used successfully for further extensive grafting

This patient could not have had a surgical correction of his lymphatic obstruction problem in any way without the employment of this technic We have performed the same procedure successfully on 41 other individuals and the operating time has decreased to three to four hours This is accomplished by using two teams working on opposite sides of the limb at the same time A cellophane pattern of the recipient area is cut and the grafts are made to fit like a skin tight trouser before being placed on the muscle

**Results** —Our total operative cases for massive lymphedema excluding the postthrombotic type now equal 64 Of these, we consider 52 excellent 6 not particularly satisfactory and 6 not improved One of our early

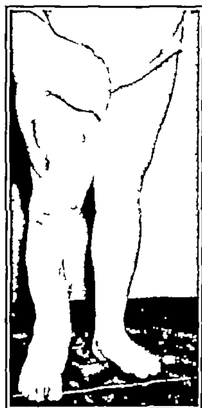


FIG 230 —Patient with massive lymphedema in Fig 229 End result. No recurrence in four years (Pratt courtesy of J A M A.)

patients died three months subsequent to all the surgical procedures from mesenteric thrombosis which was in no way related to the operation All the other patients survived and have been followed for one to fourteen years Forty-one have had the operation detailed Of these latter patients there has been no recurrence of the lymphedema.

**Other Applications of This Operating Technic.**—This type of operation has been applied to the congenital hemangiomas of the cavernous type to other congenital anomalies such as arteriovenous fistulas to patients with extensive or multiple ulcers and to patients with widespread tumor formations such as the neurofibromatas Patients with such lesions have been operated successfully with this technic



FIG 231 —Enormous section of skin subcutaneous and deep fascia being removed after denuding it of the epithelium (Pratt, courtesy of J A M A )

## LYMPHEDEMA OF THE UPPER EXTREMITIES

**Etiology.**—Theoretically, any of the causes given for lymphedema of the lower extremities can be the origin of lymphedema in the upper extremities Actually, lymphedema of the upper extremities is rare unless it is of malignant origin Only 6 out of 88 patients with lymphedema seen by the author had upper extremity involvement The most common cause of this condition is carcinoma of the breast and/or radical mastectomy The lymph gland drainage from the breast is so extensive that the retention of involved glands even after quite adequate surgery is possible That excision of the axillary glands *in toto per se* does not result in lymphedema is proven by a large number of patients so treated for breast growth who have normal arms Tumor recurrence usually causes the lymphedema which follows radical mastectomy for carcinoma of the breast. So often is this true, that when lymphedema follows carcinoma of the breast, the burden of the proof is that it is not recurrence Injuries, operations, burns, and all types of radiation therapy can cause lymphatic blocks Venous thrombosis in the axillary or subclavian vein will be followed by some degree of lymph stasis, and this lesion is the second most common cause Injury may be the cause for the lymph stasis In a few patients, a lymphedema

develops after a mastectomy or other operation in or around the axilla where there is no malignant recurrence A lymph stasis secondary to the





Fig 232 —Anterior view Preoperative and postoperative pictures of patient with massive lymphedema complicated by ulcerations and dermatitis.

operation trauma or scar then is the cause This condition, while infrequent, may occur Allergy may cause a lymph collection in the upper extremities

**Symptoms.**—The symptoms have been detailed adequately under lowe extremity lymphedema and are similar The swelling after carcinoma c the breast is of the brawny, hard, "wooden-like" type which pits bu slightly Even with recurrent carcinoma, some of the swelling will g down with elevation Radiotherapy increases the incidence, as the radia tion seals off lymphatics left after the operation

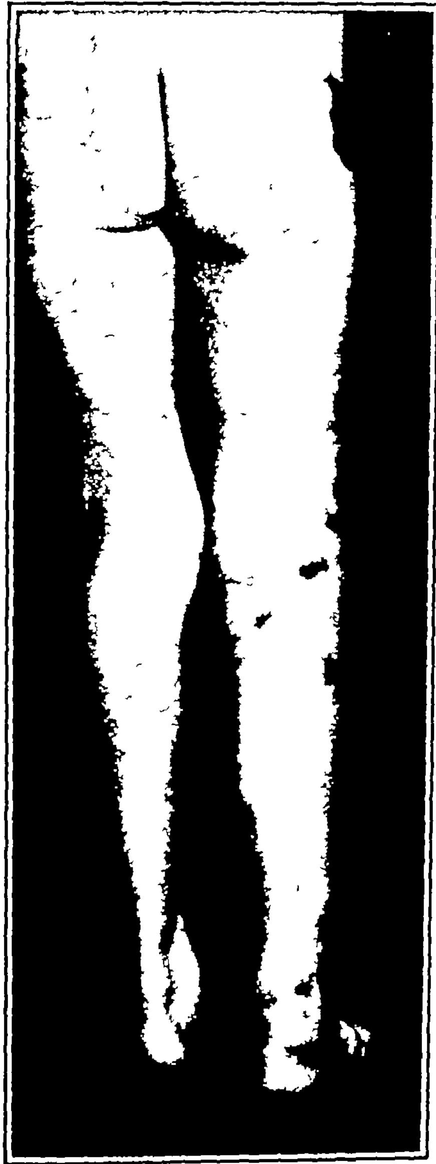


FIG 233 —Congenital hemangiomata (arteriovenous fistulas and tumors) T patients respond exceedingly well to this same operation technique

**Treatment.**—(*a*) *Prophylaxis*.—Operative intervention in the axilla should be performed carefully. The axillary vein can be thrombosed by trauma. Elevation of the arm and a compression bandage after radical axillary or supraclavicular surgery may reduce or prevent the development of a mild edema. It is most important to permit free use of the arm after operation. Nature has measures for preventing the edema if we do not interfere with her ways. In the allergic patient avoidance of the irritant, desensitization and antihistaminic drugs may help. If the cause is a venous thrombosis scalenotomy may be considered.

(*b*) *Surgical Treatment*.—The treatment of axillary and subclavian vein thrombosis has been considered on page 645. All types of operations have been attempted for lymphedema of the arm. Silk threads and silver and steel wires and needles have been placed as aids in drainage but such operations have been ineffective. In the lymphedema due to venous thrombosis an early program including antithrombotic therapy, scalenotomy, elevation, support, and block of the sympathetic system have been effective. (See pages 645 to 646.)

In the few patients who develop a true lymphedema similar to that seen in the lower extremities and where carcinoma can be excluded, an operation similar to the one described for the lower extremities can be performed. A single negative biopsy for carcinoma should not be accepted as proof that no cancer exists. One must accept the inevitable scarring and possible keloid formation. Such residual scars can be covered cosmetically or with a glove support.

Excision of congenital lymphatic collections with plastic repair is a primary object. Subsequently, the technic of grafting as described in the following paragraph applies.

In the lymphedema secondary to *x* ray or radium burn one must first exclude recurrent carcinoma. If this can be done, escharred, indurated, retracted tissue should be plastically excised and the muscular tissues mobilized. Physiotherapy at this stage may be required. Skin grafting thereafter is indicated. The recipient area may be so scarred as to delay or prevent "takes." Subsequent efforts, however, may permit healing.

In the extreme instances the same operative principles may be followed. After the removal of the epithelium at [illegible]/1000 to [illegible]/1000 of an inch thick, pathologic tissue is excised *including* the deep fascia. Subsequent reapplication of the epithelium, especially of the fingers, requires excellent plastic surgical judgment. Rehabilitation is of great importance. One must emphasize (1) the removal of only the epithelium to a depth of [illegible]/1000 of an inch, (2) excision of all the pathologic tissue including the deep fascia and (3) plastic reapplication of the epithelium.

## FEBRILE REACTIONS AFTER LYMPHEDEMA OPERATIONS

These patients' susceptibility to infection is reduced but not eliminated as their lymphatic defenses are absent. Attacks of fever may occur. Antibiotics, both prophylactically and therapeutically, should be used. These should be given prior to the expectation of an attack. A pattern of the previous attacks will indicate when these drugs should be given.

**Symptoms.**—The symptoms have been detailed adequately under lower extremity lymphedema and are similar The swelling after carcinoma of the breast is of the brawny, hard, "wooden-like" type which pits but slightly Even with recurrent carcinoma, some of the swelling will go down with elevation Radiotherapy increases the incidence, as the radiation seals off lymphatics left after the operation



FIG. 233 —Congenital hemangiomata (arteriovenous fistulas and tumors) These patients respond exceedingly well to this same operation technique

In the lymphedema due to venous thrombosis, the veins will be dilated Venograms will show the block The veins remain even after recanalization and may be of the varicose type as the valves are destroyed The venous pressure in such veins is high (5 times normal) [23] (See page 677 and chapter on venous thrombosis, axillary and subclavian, page 640 )

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In the extreme instances, the same operative principles may be followed. After the removal of the epithelium at $\frac{10}{1000}$ to $\frac{15}{1000}$ of an inch thick, pathologic tissue is excised, *including* the deep fascia. Subsequent reapplication of the epithelium, especially of the fingers, requires excellent plastic surgical judgment. Rehabilitation is of great importance. One must emphasize (1) the removal of only the epithelium to a depth of $\frac{10}{1000}$ of an inch, (2) excision of all the pathologic tissue including the deep fascia and (3) plastic reapplication of the epithelium.

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# Section VII

## Auxiliary Surgical Procedures

*Diversion of Circulation Roentgen Ray Visualization of the Cardiovascular System Skin Grafting*

## Chapter 38

## DIVERSION OF THE CIRCULATION

### *Methods of By passing Parts or Sections of the Heart and Vessels*

SINCE the time cardiac surgery first seemed feasible surgeons and physiologists have considered rerouting or by passing the circulation past the operative site Thus if the inflow from the superior and inferior venæ cavæ could be halted a dry right side of the heart would be obtained In a similar manner if the pulmonary vein and aorta flow were by passed the left side of the heart could be opened with impunity Such a mechanism would permit direct attack on intracardiac lesions of the valvular type or septal defect and allow therapy on deficient coronary vessels Normally the circulation can be halted with impunity only for three to three and one-half minutes Life is compatible with an obstructed circulation for as long as seven to ten minutes The brain however deteriorates when it is deprived of oxygen after the critical three-minute period

Shunting blood around the mitral valve has been tried experimentally by several investigators In general the method has been to use some shunt from the pulmonary vein or left auricle to the left ventricle The feasibility of inserting a valve into such a shunt has been tried These valves have been made from plastic materials and also have been supplied by homologous grafts The results have shown that a by pass at least has potential application. The introduction of a foreign or rigid material as a valve will cause inevitable pressure ulceration and eventual perforation The use of a homologous graft technically is difficult and is successful in experimental animals only in 1 out of 4 trials

A by pass of a temporary nature on the other hand is not only feasible but has been evolved This may be performed by a pump This pump may take advantage of oxygenation by an external apparatus or in other instances may utilize the patient s own pulmonary system The use of a homologous donor s pump and respiratory system likewise is under study

Some of the experimental work so far reported and at least partly successful is detailed Without question these reports will furnish leads to future achievement

**Extra Cardiac Shunts** (Experimental) —The possibilities of this operation were suggested by Jeger in 1913 [26b] He suggested that a vein shunt be made from the pulmonary vein to the left ventricle, using a vein which had intact valves In 1914 Tuffier and Carrel[43] by-passed the pulmonary valve. The plastic materials which became available in the last few years have renewed interest in such by-passes Hurwitt[23] by-passed the pulmonary valve by using a plastic tube from the right ventricle to the pulmonary artery Some of the cats used in his work survived Hufnagel[22] showed that a methyl methacrylate tube could be used in the aorta and Ingraham *et al* [24] used polyethylene tubes

**Preserved Aortic Shunt.**—Gross, *et al* [20,21] used an aorta which had been removed from a dog and preserved for days in a cold (not frozen) modified Tyrodes solution To prevent regurgitation they used a fresh aortic valve from another dog The graft was inserted from the left auricular appendage to the left ventricle To maintain the aperture in the ventrical wall, a sleeve of polyethylene encircled the aortic valve While these experiments were only partially successful, future possibilities are recognized from this work The autopsied dogs showed that the graft was patent, although considerably narrowed and fibrosed, but that in each case the grafted valve degenerates and leads to regurgitation The use of the rigid material at the site of anastomosis was shown to cause pressure, ulceration, and eventual fatal hemorrhage This is in line with previous discussions on rigid tubes These observers believe that a mechanical valve can be constructed eventually The author believes that such a valve to be successful must be of non-rigid construction.

A simple pump method of developing a dry left side of the heart was described by Clowes [7] The oxygenated blood from the left pulmonary vein is diverted into the pump which injects it into the systemic arterial circulation, a clamp is placed on the right pulmonary artery to prevent blood passing through that circuit The dry cardiac cavity will be made available for direct visual surgery in time

Many surgeons have had the idea that the circulation might be maintained entirely outside of the body Thus, the functions of the heart and lungs would be performed mechanically rather than by the patient's cardiac and respiratory systems Such a mechanical or extracorporeal system would have two main functions It might be utilized when either the heart or lung were seriously or completely impaired temporarily In addition, if such an apparatus functions satisfactorily, intra-cardiac and intra-pulmonary operations could be performed in an open manner with direct vision without hemorrhage Such apparatuses have been devised for animal use As early as 1937, Gibbon[41] had reported on the artificial maintenance of circulation during occlusion of the pulmonary artery, experimentally [41] The earlier work on perfusion experiments by Brodie,[3] Euler,[17] Bourne,[5] Drinker[16] and others, and the work on blood oxygenation by Cruickshank,[10] Daly *et al* ,[11] and others, was in this line O'Shaughnessy,[33] one of the great investigators of our era whose untimely death in World War II deprived us of a noted investigator, had perfused the brains of animals, after clamping the vena cava, with Ringer's solution, to which a 5 per cent hemoglobin had been added, and kept the animals (a cat and a dog)

alive indefinitely Crafoord [9] in 1948 repeated this work Perfusion of the entire body was a natural outgrowth of this work Such perfusion did not completely interrupt the blood flow as the venae cavae were not clamped

**Cardiac Pumps** — *Descriptions of Pumps* —Two types of pumps have been developed Those with internal valves have the disadvantage of trauma The nonvalvular type was first described by DeBakey [13] for continuous blood transfusions Three general methods have been employed

1 *Mechanical Method* —Oxygen may be mixed into the blood mechanically by the injection of air or oxygen in a bubbling method [12,28,32,35,40,45] The second mechanical method is by injecting oxygen and blood under pressure against the inside of a spherical glass bow [17] The third mechanical method is by administering oxygen intravenously without attempting to mix the blood outside the body [8,14,39,46]

2 *Chemical Method* —The chemical mixing of oxygen with the blood has been done in many ways These include the introduction of hydrogen peroxide[30,32,33] and sodium percarbonate [33] both given intravenously

3 *Oxygenation by Blood Film* —The exposure of blood to oxygen by establishing a blood film on various objects has been performed The film was placed on glass beads by Bornstein [4] on an inverted jar by Richards [38] on revolving horizontal glass cylinders by Frey [19] similar aluminum plates by Bayliss *et al* [1] and Evans *et al* [18] on cylinders by Daly [11] Jongbloed [26a] on other revolving tubes and metals [10,27] and by dripping blood through an oxygen-impregnated atmosphere by Thomas [42] Gibbon [21,41] passed oxygen over a thin film of blood on the inner surface of a revolving vertical cylinder and obtained 98 per cent of oxygen saturation Other methods included increasing the revolutions raising the amount of blood and oxygen foaming and various modifications of these procedures

**Temporary By passing of the Left Ventricle by a Pump Method** —A *Zollinger Pump* —Experimentally this by pass has been performed in dogs by Zollinger and his associates [47] A tube carries the blood from the atrium around the left ventricle by passing it through a cannula to a pump the pump thus taking the place of the ventricle This permits the animal's own lungs to oxygenate the blood This work has not been fully developed [31]

B *Gibbon Pump* — In his most recent version of the pump Gibbon[21] devised an oxygenator which consisted of 6 stainless steel screens that were suspended from a distributing chamber and enclosed in a plastic case With this chamber he produced an extracorporeal blood circuit and by-passed both the heart and lungs The venae cavae could be occluded This radiator like device supplies oxygen to the blood The pump draws blood out of the venae cavae it is oxygenated and then another pump forces the blood back into the arterial system for distribution to the body With this pump Gibbon has performed successfully an operation under direct vision on a septal defect in a human patient The blood flow through the heart was stopped and replaced by the pump for twenty four minutes This success heralds great future possibilities

C *Dennis Pump* —Dennis[12] also devised a pump which similarly produces a dry cardiac chamber Dennis pump is of the pump-oxygenator type This apparatus pumps blood from the venae cavae through a flow

meter. A jet system films the blood on rotating screen discs for the exchange of oxygen and $CO_2$. Heparin is added to prevent blood coagulation.

*Other Pumps.*—Sewell and Glenn[37] and Leeds[29] have developed pumps to circumvent the right heart, and Sirak,[39a] as well as Dodrill,[15] had worked on one to by-pass the left heart. Welch[44] has developed a pump for both sides of the heart. Gibbon[31] and Dennis[13] also attempt to replace the work of both the heart and lungs.

**Exclusion of the Heart by Parabiosis.**—Noting the difficulties of these oxygenator-pump apparatuses, Blum[2] devised an exclusion method for shunting the blood stream from the heart. He uses a companion animal as the oxygenator, maintaining the blood exchange by a double pump. This method has the disadvantage of placing a well animal (or person) in danger during the procedure. Some combination or modification of these methods may be the ultimate answer.

**Hypothermia to Permit Closure of the Inlet and Outlet of the Heart.**—It has been found that the brain can tolerate longer periods without oxygen if the patient's temperature is lowered. The length of time that the circulation can be shut off to the brain has been lengthened from three and one-half to fifteen minutes by this simple expedient.[8] This method would permit a dry right side of the heart for this time interval with occlusion of the superior and inferior venae cavae. A similar method could be used for the left side of the heart. We have used hypothermia in operations on all central aneurysms.

**Direct Diversion of the Extracardiac Circulation.**—A simple method has been used to divert the circulation past an operative point in the aorta during operations for aortic aneurysms. With two 15-gauge needles and a piece of polythene tubing a circuit of blood can be maintained around an operative point. This can be used to supply the brain by placing a needle in the aorta as it leaves the heart and then into one or both of the carotid arteries. It also is applicable to by-passing blood to the iliac arteries when an operation is being performed on the abdominal aorta. It is a simple expedient to use when there is a shut-down of the circulation through the carotid arteries. The system may have a T or Y connection so that heparin may be added as needed. This same method can be used on other vessels besides the aorta.

**Simple Pump Diversion of Circulation.**—It has been proven that many simple methods of shunting blood past a certain point in the vascular system work well, especially in these parts distal to the heart itself. We have utilized one of the old "direct blood transfusion apparatus" machines as a satisfactory mechanism to pass blood along the vascular tree. The blood transfusion apparatus originally devised by Soresi aspirated 2 cc of blood at a time and injected it immediately. In our hands in the days of the direct blood transfusion this apparatus was the most effective one. We have used this same machine, with the addition of an electric pump, to by-pass an obstructed point. In the lower extremity, about 2 cc of blood normally enter the arterial tree with each systole of the heart. Gauging our machine on this figure we feel we can reproduce the heart action locally by such an apparatus. This permits shutting off the circulation to a part for a long time. It also permits us to know if the circulation distal to

the operative site is working satisfactorily. It avoids distal clotting. It has been used in the surgical treatment of aneurysms particularly.

**Vein Grafts to Divert Circulation.**—This method has been utilized in the circulation beyond the heart itself innumerable times. The use of an analogous vein has the advantage of not jeopardizing some other part of the body as occurs when an analogous arterial graft is used. It also eliminates the dangers inherent in the use of a foreign material such as an homologous graft. See pages 340 to 341.

**Homologous Grafts to Divert the Circulation.**—The indications for the use of homologous grafts to reconstruct or correct the arterial circulation have been described. These grafts, their procurement, storage, and the technic of their application is the same as outlined on page 341 and needs no repetition.

Thus our efforts to create an artificial heart extracorporeally or to divert circulation from a section of the heart to make a dry field have been started but have not succeeded in a practical way. The need for further work in this line is recognized. While there are many physiologic and hematologic problems, at times it appears that the mechanical ones are the farthest from solution. The great progress made in tool production would make it appear that the utilization of the mechanical brain which evolves these efficient apparatuses may be of future use. Some combination of such minds with biophysic and physiologic insight may solve this problem. The diversion of the circulation around and distal to the heart is not impossible and requires only refinement of the technic.

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**Simple Pump Diversion of Circulation.**—It has been proven that many simple methods of shunting blood past a certain point in the vascular system work well, especially in these parts distal to the heart itself We have utilized one of the old "direct blood transfusion apparatus" machines as a satisfactory mechanism to pass blood along the vascular tree The blood transfusion apparatus originally devised by Soresi aspirated 2 cc of blood at a time and injected it immediately In our hands in the days of the direct blood transfusion this apparatus was the most effective one We have used this same machine, with the addition of an electric pump, to by-pass an obstructed point In the lower extremity, about 2 cc of blood normally enter the arterial tree with each systole of the heart Gauging our machine on this figure we feel we can reproduce the heart action locally by such an apparatus This permits shutting off the circulation to a part for a long time It also permits us to know if the circulation distal to

the operative site is working satisfactorily. It avoids distal clotting. It has been used in the surgical treatment of aneurysms particularly.

**Vein Grafts to Divert Circulation.**—This method has been utilized in the circulation beyond the heart itself innumerable times. The use of an analogous vein has the advantage of not jeopardizing some other part of the body as occurs when an analogous arterial graft is used. It also eliminates the dangers inherent in the use of a foreign material such as an homologous graft. See pages 340 to 341.

**Homologous Grafts to Divert the Circulation.**—The indications for the use of homologous grafts to reconstruct or correct the arterial circulation have been described. These grafts, their procurement, storage, and the technic of their application is the same as outlined on page 341 and needs no repetition.

Thus our efforts to create an artificial heart extracorporeally, or to divert circulation from a section of the heart to make a dry field, have been started but have not succeeded in a practical way. The need for further work in this line is recognized. While there are many physiologic and hematologic problems, at times it appears that the mechanical ones are the farthest from solution. The great progress made in tool production would make it appear that the utilization of the mechanical brain which evolves these efficient apparatuses may be of future use. Some combination of such minds with biophysic and physiologic insight may solve this problem. The diversion of the circulation around and distal to the heart is not impossible and requires only refinement of the technic.

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# Chapter 39

# ROENTGEN RAY VISUALIZATION OF THE CARDIOVASCULAR SYSTEM (BY THE INJECTION OF OPAQUE MATERIAL)

## *Aortography, Arteriography, Venography, Angiocardiography, Cardiovascular Catheterization, Splenic and Portal Portagram*

THE technic and the skill of the cardiovascular surgeon and roentgenologist in cardiovascular visualization have so advanced in recent years that this medium has become of great value in the study and therapy of lesions of the cardiovascular system This field has become so important it is now a diagnostic, prognostic and a therapeutic subdivision of cardiovascular surgery These specialists must be skilled in roentgen ray technics and diagnosis and they must have a thorough knowledge of the cardiovascular anatomy They must know well the normal and pathologic physiology of the cardiorespiratory system and be able to correlate the results of their studies with the clinical findings in the patient To this must be added the ability to place needles in arteries and veins, pass catheters through vessels and chambers of the heart and to time correctly the exposure following a dye injection Another advance has consisted in the development of rapid serial roentgenograms Originally, we were able to obtain one, or at best, two pictures Now we can obtain two films per second X-ray films are available in the form of a continuous roll, 75 feet in length and 9½ inches wide Such exposures are like motion pictures

Contrast media and radiopaque substances may be used

1 To diagnose cardiac anomalies, congenital defects, such as patent ductus arteriosus, coarctation of the aorta, pulmonary stenosis, mediastinal and other tumors and help to prepare the surgical attack on such problems as are amenable to operation. Angiocardiography permits the study of the chambers of the heart and the associated great blood vessels as they are visualized by an intravenous injection of a radiopaque solution Over 10,000 such examinations have been made [8]

2 To determine the extent and degree of calcification as well as the collateral circulation in obstructed or partially obstructed blood vessels such as occur in arteriosclerosis

3 To obtain a better concept of the course and patency of the blood vessels or the collateral circulation in the following instances. (a) arteriovenous fistulas, (b) arterial aneurysms, where it is necessary to know if the collateral circulation is sufficient to permit obliteration of the sac; (c) congenital vascular lesions, (d) occlusive diseases with injuries or emboli in arteries

4 To ascertain the status of the venous circulatory return preparatory to surgery For example in subclavian or axillary vein thrombosis it must be determined whether the original site of thrombosis has recanalized or whether the collateral circulation is satisfactory The adequacy of the femoral venous return must be known before superficial veins are ligated

5 To visualize other organs especially the splenic and portal circulation and outline abnormalities or tumors

The possibilities of angiographic and angiocardiographic visualization are unlimited The circulation of any organ may be determined satisfactorily by arteriograms with the dye being injected above or directly at the site at which the blood vessel enters the organ Farinas[12] demonstrated the circulation of the kidney and other organs ten years ago

## VASCULAR VISUALIZATION OF ORGANS

Angiocardiography in spontaneous and artificial pneumothorax has demonstrated avascularity in tuberculous lesions displacement of pulmonary blood vessels shifts of the heart great vessels and mediastinum decrease in the vascularity of the collapsed lung and delay in the rate of fresh blood flow[22] Lung tumors and the operability of cancer have been determined in a similar fashion The demonstration of involvement of the vascular tree by new growth indicates inoperability[7, 17] At present this must be confirmed surgically The early differentiation of aortitis of the syphilitic type before clinical proof exists has been utilized for earlier therapy The signs are a dilatation (over 38 mm. in the mid ascending aorta) irregularity and tortuosity variations in the wall thickness frequent calcification of the ascending aorta and aneurysm[21] Emphysema fibrosis and mediastinal tumors have been demonstrated by this method[8] Angiography or aortography have determined aneurysms renal tumors[18] renal adequacy all types of occlusions[8, 11, 15, 19] and the blood supply to such organs as the spleen and liver,[26] the uterus the adrenals and even the human placenta[13] The saphenous vein route has been used for visualization where the superior vena cava was blocked[14]

*The Length of the Aorta* —The length of the aorta under normal and diseased conditions is of value diagnostically and therapeutically By angiocardiographic measurements it has been found that the thoracic aorta can be determined by the following formula length in cm equals 6 76 plus 15 86 $\log_{10}$ age The normal variations from this curve show a standard deviation from the mean length of ±5 07 cm. The conditions which cause aortic elongation are age arteriosclerosis syphilis and hypertension Syphilis causes elongation and dilatation of the ascending aorta which is diagnostic from arteriosclerosis or the aorta enlargement due to hypertension

X ray visualization of the chest at appropriate intervals following the rapid intravenous injection of a radiopaque substance permits visualization of the vascular tree The innominate vein the vena cava the right atrium and ventricle the pulmonary artery and its branches and subsequently the pulmonary veins the left atrium and ventricle and the thoracic aorta can be demonstrated[29]

To this method has been added *cardiac catheterization* and the knowledge that has followed this procedure. The technic is not without danger, and its interpretation must be made by one who is not only an excellent roentgenologist and anatomist but who also has a thorough knowledge of the vascular system. This modality permits a catheter to be passed through normal and abnormal valves and past septal defects. Specimens of blood can be drawn, and studies of their oxygen content and the pressure of the blood are valuable in ascertaining whether this blood is of normal content and pressure for the chamber from which it was drawn.



FIG 234.—Aortogram demonstrating aneurysmal dilatation of iliac artery after ligation of femoral artery for ruptured aneurysm. (Courtesy Dr Philip Waldman.)

**Dye and Method.**—1. *Aortogram.*—The patient lies on the x-ray cassette. The aorta may be injected by direct paralumbar puncture, the needle being inserted posteriorly from the left side. Its visualization is used most often for the lumbar aorta, the iliac and femoral arteries. With some experience the needle can be inserted just below the renal arteries. These arteries arise from either side of the aorta directly below the superior mesenteric artery. The left one is higher than the right. Once the needle



Fig 235 —Aortogram showing advanced arteriosclerosis in female aged thirty-six. Right iliac artery segmentally obstructed. Left external iliac artery occluded blood supply through hypogastric artery Note extensive collateral blood supply Entire arterial supply of the pelvis including the uterus has been delineated.

is in place the vessel is injected with normal saline The dye is injected best by a pressure machine modified after the technic of Dos Santos, Milanes Lopez etc. Fifty cc of 70 per cent diodrast is injected through an 18-gauge needle with the pressure of approximately 30 pounds in the injection machine. The $x$ ray is taken at five to six seconds Usually only one picture is necessary, but serial studies with the Fairchild machine

can be taken The vessel is flushed with saline immediately after the dye injection The injection can be made without anesthesia

2 *Arteriogram* —The artery to be visualized is injected above the site of pathology For the lower extremities, the common femoral artery can be approached just below the inguinal ligament where it is situated sub-



Fig 236 —Ulnar artery arteriogram showing congenital arteriovenous fistulas Note innumerable communications and accumulation in venous pools (Pratt, courtesy of *Surg , Gynec and Obst* )

cutaneously When the needle enters the arterial lumen, there will be a squirting of arterial blood into the syringe synchronous with cardiac systole Fifteen cc of 30 per cent Urokon* (sodium acetrizoate) is injected For leg artery visualization, the picture is taken in four to five seconds. Where there is an arterial block, this may be delayed from six to eight or even as long as twenty seconds

* Mallinckrodt Chem Co , St Louis

With rare exceptions it is not necessary to make an incision to expose the blood vessel In some of the smaller vessels or where there is calcification this exposure may be required In such instances the control of the dye can be maintained with a blood pressure cuff

Retrograde technic includes the introduction of a needle into the artery Through this needle then is passed a polythene tube which extends proximally or distally depending on the part of the body's blood supply one desires to see in the picture This tube is guided by a fluoroscope until it is in the exact place The dye then is injected This is best obtained by the injection of dye under pressure Ten to thirty pounds pressure will



Fig 237 —Arterial circulation hand—Thorotrast injected Visualization of palmar arches and collateral circulation

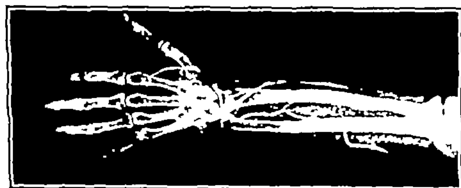


Fig 238 —Same patient after dye has entered the venous system Note venous valves well demonstrated

concentrate the dye at the desired point Simultaneously the picture is taken Sometimes the application of pressure cuffs below the point at which the picture is to be taken further maintains the concentration of the dye in the area

The technic of entering an artery with a needle requires experience. The feel of the needle against an artery the amount of pressure necessary to enter it and the depth of the various vessels involved can be learned best on a fresh cadaver

The routine surgical exposure of major blood vessels for arteriograms is not needed as shown by the excellent results of Wright [28] who reported that he had to expose the artery only once in approximately 400 arteriograms

3. *Venogram*.—With the improvement in the technic and timing, the previous difficulties in the interpretation of venograms in the lower extremities have been eliminated. The status of the deep veins as to valve competency can be determined adequately. The points at which communicating valves fail can be visualized and this directs the surgical therapy. In several instances, a failing deep vein circulation has been identified prior to a contemplated superficial vein stripping. Serious disability may result if such surgery is performed. The veins most often

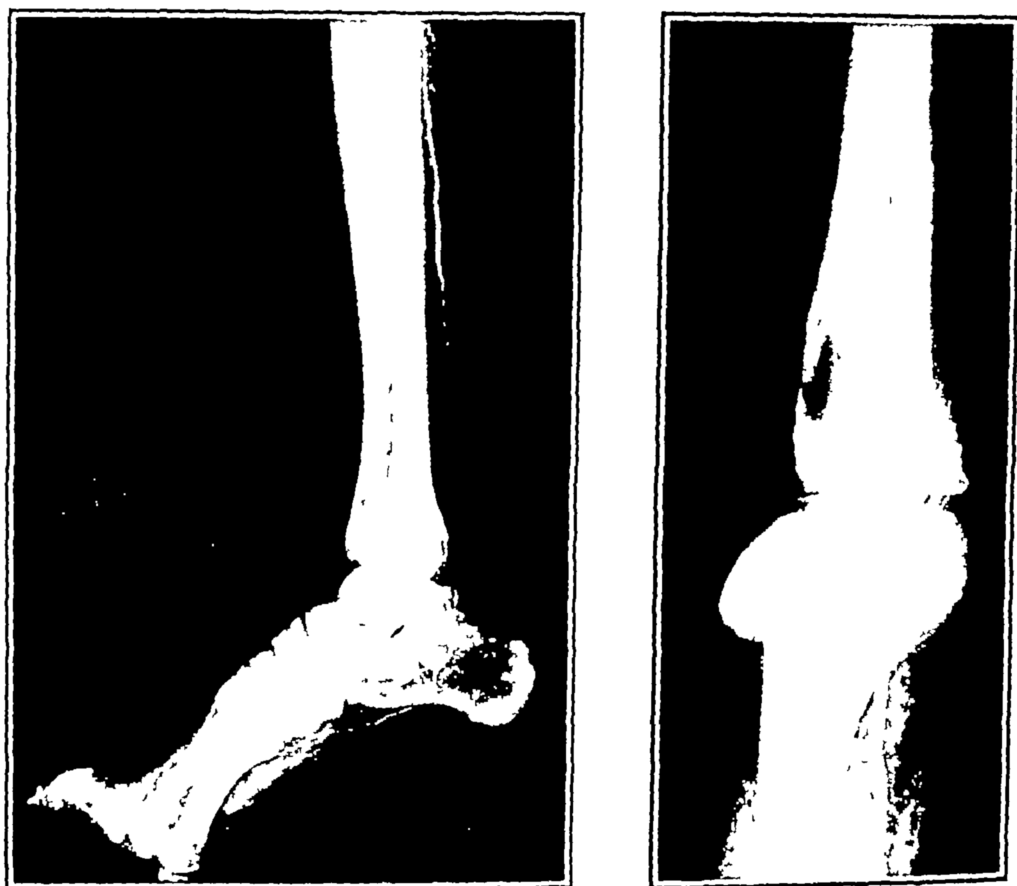


Fig. 239.—Arteriogram—femoral artery. Note mouse-eaten appearance of artery, indicating occlusive disease and its obliteration in the dorsalis pedis artery. Evidence of stimulated collateral circulation. (Courtesy of Doctors Henry K. Taylor and Teresa McGovern.)

visualized are those of the lower extremities. For this picture, 20 cc. of 30 per cent Urokon is used. If the deep veins are to be visualized, a tourniquet is placed at the ankle and the injection made in a superficial vein over the dorsum of the foot. The x-ray is taken approximately twenty-five seconds after the injection.

Information regarding the status of the veins can be obtained after arteriography if the film is taken within a few seconds after the dye is injected in the artery. By that time the dye is in the veins.

The status of the deep vein circulation can be determined also at operation time. This is important in ligating superficial veins where the deep

vein circulation is in question Dye can be injected into the common femoral vein The table can be tilted with the feet down If the dye runs into the superficial femoral vein against the valve system it indicates that the valves in this vein are defective and incompetent In the presence of an adequate femoral profunda vein this superficial femoral vein then may need resection A similar defect in the profunda or saphenous systems may



Fig 240 — Cardiography 1 second after injection Solution in the superior vena cava with the right auricle and right ventricle and pulmonary veins visualized and some solution already in left heart (Courtesy of Dr H K. Taylor)

be shown It should be emphasized that the *dye should not be injected into a recently inflamed or thrombosed vessel*

4. *Angiocardiogram* — The technic of angiocardiogram includes the injection of 50 cc of 70 per cent diodrast the entire injection being completed in one second Films are taken at one-half to one second intervals with the Fairchild type of multiple pictures By the ninth second the left ventricle is visible and in eleven seconds the dye is in the aorta

*Technic of Angiocardiography*—The patient omits the preceding m
A sterile tray with the standard supplies for an intravenous injectio
prepared Control roentgenograms are taken A small amount of a
thesia is placed over a suitable elbow vein The needle is then introdu
and checked by normal saline A circulation time test can be perform
This is directly or indirectly done by the injection of 5 cc of a 20 per c



Fig 241 —Cardiography, 4 5 seconds after injection (Courtesy of Dr H K Taylor)

solution of sodium dehydrochlorate At the time of injection, a stop watch is begun and this is stopped at the exact moment the patient notices a bitter taste in his mouth This will help in determining the time to make the exposure The contrast substance is then attached to the syringe With the machine rotating, the patient is instructed to exhale deeply The patient then inhales The following events then take place at the same moment As the patient inhales, the doctor injects the solution and the

technician starts his stop watch At the end of the deep inhalation the patient holds his breath until the first exposure has been made The other exposures are made with the patient holding his breath at the appropriate times predetermined by the doctor in charge Usually the picture is taken every one-half second for ten seconds

The needle is left *in situ* while the films are developed It is then removed if the films are satisfactory

*Other Dyes* —Thorotrast (stabilized barium dioxide solution) has been used for the visualization of large blood vessels Five cc of the solution are sufficient for the upper extremities but 12 cc are needed for the lower extremity vessels This substance is radioactive and is eliminated through the liver and kidneys The possibility of delayed radiation burns therefore exists At the present time it is used in our Clinic only for the visualization of the cerebral vessels

Skiodan neo-iopax and sodium iodide the latter in strengths of 30 to 70 per cent (in some instances up to 100 per cent) have certain local and general reactions They have been replaced by safer substances

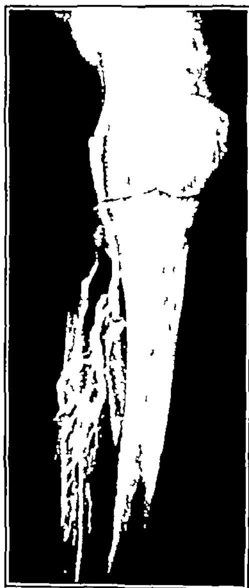


Fig 242 —Venogram illustrating the importance of determination of deep vein circulation. Pathology below the knee due to incompetencies of perforators. Saphenous vein adequate and essential in the thigh

*Cardiac Catheterization* —Cardiac catheterization is an additional diagnostic procedure which has its accepted place in cardiophysiological studies It is not without danger and therefore should be used only when additional information to confirm a diagnosis or to determine a therapeutic procedure is required Skilled interpretation of the catheterization findings is of the utmost importance

*Method* —A radiopaque catheter similar to a ureter type with a built in curved tip in sizes 6 to 9 is used Its purpose is to record pressures in various parts of the heart and great vessels to withdraw blood samples in similar areas and to pass through or determine the presence of septal defects Using a vein, data on the right side of the heart can be deter-

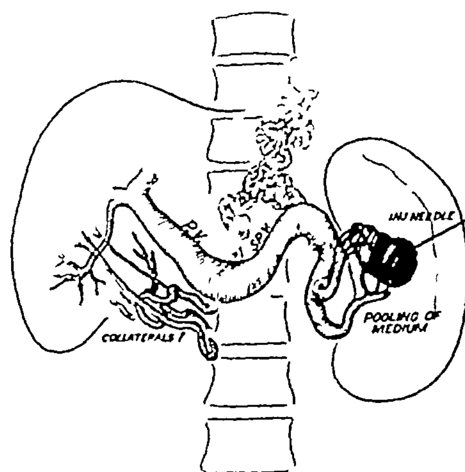
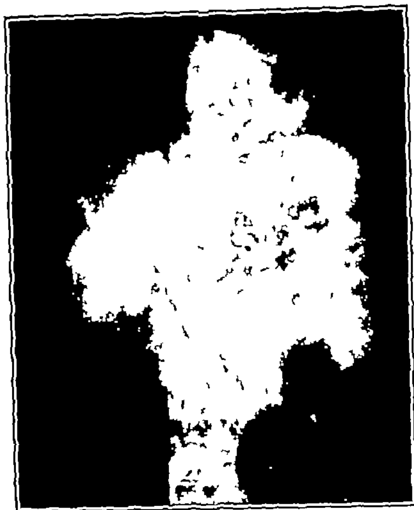



FIG 242a —Portal hypertension in cirrhosis of liver with hypersplenism Splenic portagram tripod type Collateralization by coronary vein (Note varices in lower esophagus and cardia of stomach and on the right side inferior to the portal vein) These may anastomose with the liver veins directly or communicate retroperitoneally with the inferior vena cava Large diameter of splenic vein makes it usable for splenorenal shunt performed following splenectomy (Courtesy Drs Rousselot, Ruzicka and Doehner and permission of *Surgery*)



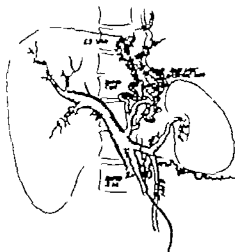


Fig. 242b.—Portal hypertension in cirrhosis of liver. Portal portagram. Superior mesenteric vein cannulized at surgery. (Note diffuse density representing residual dye in splenic area from splenic portagram.) Dye flow in portal system is hepatofugal over two chief collaterals, the coronary and the inferior mesenteric veins. Submucosal gastric varices in the cardiac region of the stomach and esophageal varices in distal esophagus. Hemiazygos vein is also visualized. Because splenic vein is below 1.0 cm. in diameter and portal vein is large, portacaval shunt was used rather than splenorenal shunt. (Courtesy of Drs. Rousselot, Ruzicka and Doehner and permission of *Surgery*.)

mined. Therefore its most practical application has been in the diagnosis of congenital heart disease. The catheter is inserted in the median basilic vein after exposing such vein directly. A saline infusion set keeps the catheter filled and prevents clotting. The catheter is guided under fluoroscopy control through the innominate vein into the superior vena cava. It is then advanced into the right auricle, the right ventricle and either pulmonary artery. It can be extended to the small pulmonary radicles. Blood samples are taken successively from the main branch of the pulmonary artery, the main trunk of the pulmonary artery, the right ventricle at its outflow, the right ventricle near the tricuspid valve, the right atrium near the tricuspid valve, the upper and lower parts of the right atrium and the superior vena cava. The blood specimens are taken under sterile mineral oil to avoid contact with room air. These samples are analyzed for their oxygen content by the Van Slyke method. The patient's oxygen consumption is determined by collecting his expired air for three minutes. The oxygen content of this air and that of room air (as determined by the Haldand apparatus) represents the patient's oxygen consumption. The cardiac output is determined by the Fick principle.

THE FICK PRINCIPLE

$$\text{Cardiac output} = \frac{\text{Oxygen consumption (cc./min.)}}{\text{Arteriovenous } O_2 \text{ difference (cc./l.)}}$$

$$\text{Arteriovenous } O_2 \text{ difference} = \text{oxygen content in femoral artery blood minus oxygen content in pulmonary artery blood}$$

The cardiac output is determined during exercise as well as at rest. A standard index is necessary for comparison purposes. The patient's body surface is divided into the cardiac output which gives the patient's cardiac index. The cardiac output for heart beat determines the stroke output. This is computed by dividing the cardiac output by the pulse rate. Thus we have the stroke output at rest and during exercise. As the catheter is withdrawn, continuous pressure tracings are made. The pressure manometer may record and also have a condenser microphone to amplify the tone. Two pressure tracings are obtained. These pressure readings are interpreted by later study. In addition, endocardio-electrocardiograms can be recorded. The catheter can become also an exploring instrument. Its passage through to the left side of the heart will indicate septal defects or malformation. It can be also directed down the inferior vena cava to the renal vein, hepatic vein or coronary sinus.

**Dangers of Intravascular Visualization Methods.**—1. *Angiocardiography.*—Twenty-six deaths in 6,824 examinations were reported up to 1949 in North America, Great Britain and Sweden. In the United States and Canada there were 18 deaths in 5,961 studies. Three of these deaths were not related to the injection. Twenty-one of the 26 deaths were in patients with congenital heart disease, 17 of these were cyanotic, the same number were under eight years of age, all with congenital heart disease, 5 were seriously ill and 3 were Mongolian idiots. A general anesthetic was

used in 16 The breakdown on the causes of death reported was incomplete It appears however that there was only one death of a patient with a healthy heart and that patient had renal disease One notes the number of deaths in cyanotic children to whose embarrassed cardiorespiratory status a general anesthetic was added Premedication with all types of drugs, such as morphine and the barbiturates was used The relative safety of the procedure in experienced hands seems assured The anesthesia problem is a factor upon which success may depend The modern anesthesiologist well recognizes the danger of too much anesthesia and inadequate aeration

2 *Aortography* —The relative impunity with which one may insert a needle into the aorta and inject directly or secondarily to catheterization has been demonstrated repeatedly The technic should be learned on the cadaver and is not a procedure for the inexperienced With care the information obtained far outweighs the potential dangers The possibility of sensitivity to dye exists Sensitization tests may help to some extent

3 *Peripheral Arteriography* —There exists a possibility that the needle might elevate a plaque or cause a local thrombosis This occurrence has been rare. Where the information is of such prognostic and therapeutic importance as to accept this slight risk the procedure always is indicated There have been no deaths and no incidences of gangrene in injections of over 500 patients in our Clinic

4 *Venography* —One should not inject the inflamed veins A phlebitis or embolic phenomena may be excited if a clotted vein is injected The possibility of allergic reactions to the dye is present Where the information to be determined from the procedure justifies a slight risk of complications no other contraindication to the venography exists

5 *Cardiac Catheterization.*—The dangers are in inverse proportion to the technical skill of the one performing the catheterization Even in experienced hands however danger exists It is selected therefore only when the data it may supply are required for diagnosis or treatment The dangers are (*a*) Rhythm disturbances—these develop when the catheter passes through the right ventricle They disappear usually when the catheter is withdrawn or advanced into the pulmonary artery Persistence of bundle-branch block or ventricular tachycardia may be serious The greatest hazard is ventricular fibrillation or standstill. This danger occurs most often in the diseased or ailing myocardium the coronary or rheumatic heart disease patients or those with irritable hearts

(*b*) Auricular disturbances—these are more rare Ectopic beats tachycardia or fibrillation have occurred Removal of the catheter from that site usually terminates such phenomena

(*c*) Embolization—air emboli should be preventable by a closed technic Dislodgement of other emboli or clot formations have followed such manipulation They are most common in patients with auricular fibrillation

(*d*) Infection—is potentially a complication but is rare

(*e*) Venous spasm—has followed the introduction of the catheter It may interfere with the procedure

mined Therefore its most practical application has been in the diagnosis of congenital heart disease The catheter is inserted in the median basilic vein after exposing such vein directly A saline infusion set keeps the catheter filled and prevents clotting The catheter is guided under fluoroscopy control through the innominate vein into the superior vena cava It is then advanced into the right auricle, the right ventricle and either pulmonary artery It can be extended to the small pulmonary radicles Blood samples are taken successively from the main branch of the pulmonary artery, the main trunk of the pulmonary artery, the right ventricle at its outflow, the right ventricle near the tricuspid valve, the right atrium near the tricuspid valve, the upper and lower parts of the right atrium and the superior vena cava The blood specimens are taken under sterile mineral oil to avoid contact with room air These samples are analyzed for their oxygen content by the Van Slyke method The patient's oxygen consumption is determined by collecting his expired air for three minutes The oxygen content of this air and that of room air (as determined by the Haldand apparatus) represents the patient's oxygen consumption The cardiac output is determined by the Fick principle

## THE FICK PRINCIPLE

$$\text{Cardiac output} = \frac{\text{Oxygen consumption (cc/min)}}{\text{Arteriovenous } O_2 \text{ difference (cc/l)}}$$

Arteriovenous $O_2$ difference = oxygen content in femoral artery blood
minus
oxygen content in pulmonary artery blood

The cardiac output is determined during exercise as well as at rest A standard index is necessary for comparison purposes The patient's body surface is divided into the cardiac output which gives the patient's cardiac index The cardiac output for heart beat determines the stroke output This is computed by dividing the cardiac output by the pulse rate Thus we have the stroke output at rest and during exercise As the catheter is withdrawn, continuous pressure tracings are made The pressure manometer may record and also have a condenser microphone to amplify the tone Two pressure tracings are obtained These pressure readings are interpreted by later study In addition, endocardio-electrocardiograms can be recorded The catheter can become also an exploring instrument Its passage through to the left side of the heart will indicate septal defects or malformation It can be also directed down the inferior vena cava to the renal vein, hepatic vein or coronary sinus

**Dangers of Intravascular Visualization Methods**—1 *Angiocardiography*—Twenty-six deaths in 6,824 examinations were reported up to 1949 in North America, Great Britain and Sweden [6] In the United States and Canada there were 18 deaths in 5,961 studies Three of these deaths were not related to the injection Twenty-one of the 26 deaths were in patients with congenital heart disease, 17 of these were cyanotic, the same number were under eight years of age, all with congenital heart disease, 5 were seriously ill and 3 were Mongolian idiots A general anesthetic was

used in 16. The breakdown on the causes of death reported was incomplete. It appears however that there was only one death of a patient with a healthy heart and that patient had renal disease. One notes the number of deaths in cyanotic children to whose embarrassed cardiorespiratory status a general anesthetic was added. Premedication with all types of drugs, such as morphine and the barbiturates, was used. The relative safety of the procedure in experienced hands seems assured. The anesthesia problem is a factor upon which success may depend. The modern anesthesiologist well recognizes the danger of too much anesthesia and inadequate aeration.

2. *Aortography.*—The relative impunity with which one may insert a needle into the aorta and inject directly or secondarily to catheterization has been demonstrated repeatedly. The technic should be learned on the cadaver and is not a procedure for the inexperienced. With care the information obtained far outweighs the potential dangers. The possibility of sensitivity to dye exists. Sensitization tests may help to some extent.

3. *Peripheral Arteriography.*—There exists a possibility that the needle might elevate a plaque or cause a local thrombosis. This occurrence has been rare. Where the information is of such prognostic and therapeutic importance as to accept this slight risk, the procedure always is indicated. There have been no deaths and no incidences of gangrene in injections of over 500 patients in our Clinic.

4. *Venography.*—One should not inject the inflamed veins. A phlebitis or embolic phenomena may be excited if a clotted vein is injected. The possibility of allergic reactions to the dye is present. Where the information to be determined from the procedure justifies a slight risk of complications, no other contraindication to the venography exists.

5. *Cardiac Catheterization.*—The dangers are in inverse proportion to the technical skill of the one performing the catheterization. Even in experienced hands, however, danger exists. It is selected therefore only when the data it may supply are required for diagnosis or treatment. The dangers are: (*a*) Rhythm disturbances—these develop when the catheter passes through the right ventricle. They disappear usually when the catheter is withdrawn or advanced into the pulmonary artery. Persistence of bundle-branch block or ventricular tachycardia may be serious. The greatest hazard is ventricular fibrillation or standstill. This danger occurs most often in the diseased or ailing myocardium, the coronary or rheumatic heart disease patients or those with irritable hearts.

(*b*) Auricular disturbances—these are more rare. Ectopic beats, tachycardia or fibrillation have occurred. Removal of the catheter from that site usually terminates such phenomena.

(*c*) Embolization—air emboli should be preventable by a closed technic. Dislodgement of other emboli or clot formations have followed such manipulation. They are most common in patients with auricular fibrillation.

(*d*) Infection—is potentially a complication but is rare.

(*e*) Venous spasm—has followed the introduction of the catheter. It may interfere with the procedure.

*Precautions* —To minimize the dangers a direct reading electrocar graphic machine of one type or another should be used constantly. T tip of the catheter should not remain in the right ventricle any longer th required. If an arrhythmia develops, the catheter should be withdra from the ventricle If these do not disappear, the procedure should terminated. If the patient becomes uncomfortable for no reason, catheterization should be discontinued. There should be facilities availa to control any circulatory emergency including cardiac arrest T fluoroscopic exposure should be kept within safe limits

6 *Splenic and Portal Portagram (X-ray Visualization of the Splenic a Portal Venous System)* —In diseases of the spleen and liver, the ven drainage of these organs is of great diagnostic and therapeutic importan The differentiation between extra- and intrahepatic blocks may be det mined and this may dictate the type of operation to be performed T spleen can be injected directly, the dye being quickly visualized in t splenic vein and its collateral branches To some extent, the size a pathological picture of the spleen is thus determined

TECHNIC OF SPLENIC PORTAGRAM —The spleen is injected from the le lower thoracic wall at the 9th interspace If the spleen is of normal size, t injection is made at the midaxillary line When the spleen is very lar the injection point is at the anterior axillary line An 18-gauge need attached to a 50 cc syringe containing 70 per cent Urokon is used Th needle is pushed into the spleen until a free flow of blood is obtained aspiration The patient is asked to hold his breath The dye is the injected as forcibly as possible The *x*-ray picture is taken at the tim that 40 cc of the solution have been injected. The *x*-ray film is center at the xiphoid process (See Figure 242a )

The collateral circulation in portal system blocks becomes extreme important The dilatation of the coronary and gastric veins is extreme significant since these lead to the varices and hemorrhages which requi surgical intervention Lack of control of this complication is a commo cause for death in portal hypertension The roentgen picture of th collateral circulation will help select the operation to be performe Whether a splenorenal or portacaval shunt should be done may be de termined at times by such *x*-rays (See Fig 242a )

TECHNIC OF PORTAL PORTAGRAM —As soon as the peritoneum is opene one of the upper jejunal bowel loops is selected A large vein close to th mesenteric vein is incised and a No 15 polyethylene catheter is tied into it Venous pressures are then taken Fifty cc of 50 per cent Urokon are the injected into this vessel with pressure The *x*-ray picture is taken afte 40 cc have been injected The *x*-ray film should be centered directly unde the xiphoid process (See Fig 242b )

## LEFT-SIDED CARDIAC CATHETERIZATION

The left heart has not been subjected to study in this way due to the danger of passing a catheter through the aortic valves It has been done in animals and probably can be accomplished in humans The techni

would have to be developed on cadavers The added risk would reserve such methods for the occasional patient in whom the information to be derived would be great enough to justify the risk

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Chapter

# 40

## SKIN GRAFTS FOR VASCULAR DISEASES

### *Types of Grafts, Preparation and Aftercare Homologous Grafts Skin Banks*

THE loss and replacement of the epithelial covering of the body is an important and never-ending part of surgical therapy of many lesions. In diseases or injuries of parts of the vascular system the success or failure of the reparative process may depend on the satisfactory replacement of lost skin. Thus every vascular surgeon necessarily must be trained in this plastic procedure and understand its essential basic principles. In the lesions of the venous system, for example, ulceration results in skin loss in approximately one-third of the patients. The therapy of lymphedema succeeds or not with the success or failure of the grafting operation. The congenital and acquired hemangiomata and aneurysms frequently require the sacrifice of large amounts of skin. The possibility of skin as a carrier of blood supply to the deficient myocardium and other avascular areas is under study. If an ulcer has been present for a long time with healing and repeated breakdowns, the epithelium has been digested away, the edges are scarred, and one cannot expect the epithelium to grow in from the edge of the ulcer and cover the base. The ulcer is similar in this way to a burn where the epithelial germinal centers have been destroyed.

After the causative pathology has been eliminated as far as it is possible, the necessity for a skin graft must be determined. In general, if the ulcer is 2½ cm in diameter, it is best to apply a skin graft because even if epithelization or scarring occurs, the surface will be so thin and friable that a minor trauma often reopens the lesions and starts the whole process over.

In patients who have been hurt at work or who have a disability covered by insurance, the replacement of the skin which is not too healthy may be most important. Many patients in this category are not too anxious to return to work. The presence of an insecurely healed skin may delay their rehabilitation further than is necessary. A healthy covering of the injured area, on the other hand, has both a physical and psychologic beneficial effect.

Most ulcers of the arterial type have an insufficient basic circulation for a graft to take. Each ulcer therefore must be treated individually.

**Preoperative Preparation of the Recipient Site.**—1. *Antibiotic Therapy.*—Prior to skin grafting all ulcers should be cultured and the specific antibiotic for the organism present should be prescribed. The drug should be given both generally and locally. It has been shown that in three of

after the local application of penicillin solution in the strength of 1,000,000 units to 250 cc of saline, streptococci no longer can be cultured from ulcers. The frequent contamination caused by *Pseudomonas aeruginosa* (Bacillus pyocyaneus) may be eliminated by the application of 1 per cent acetic acid solution for one week.

2. *Fungicide.*—Most ulcers are contaminated with some type of fungus infection, even though the fungus cannot be cultured or identified. For this reason, the use of some mild fungicide, such as potassium permanganate in 1:15,000 to 1:25,000 strength, applied twenty minutes once a day, will control such growth. Undecylinic acid-zinc undecylenate has fungicidal effect with minimal tissue irritation.

3. *Exposure of the Ulcer.*—In the experience of our Clinic, the recipient site can be prepared for grafting most rapidly by the removal of all dressings and ointments and the exposure of the area to air at room temperature. A cradle can prevent contamination of the wound by bed clothing. A saline soak will remove any serum, exudates or purulent collections. In venous type ulcers, the legs should be elevated at these times.

4. *Saline Soaks.*—A sterile, saline soak for one hour once or twice a day will help remove any discharge or drainage from the ulcer site. This also stimulates the local circulation. It is innocuous and will not irritate the tissues.

5. *Muscle Adenylic Acid*[18,19] (*My-B-Den**).—It has been found that in many chronic ulcers there is a deficiency of adenylic acid in the tissue cells. This substance is a normal constituent of cells. Its absence appears to be related to failure to heal or to delayed healing. Pruritus is controlled. In a series of experiments we were able to heal ulcers with this substance alone. Maintenance of this healing requires treatment of the underlying pathology. The substance can be given by mouth but acts best when injected in a gel which is liquid at body temperature and which prolongs its absorption rate. It is injected intramuscularly, 1 cc every second day. In only one patient has there been any allergic reactions, and in this one, dermatitis was the only response. The associated irritation disappears as does the irritation caused by the scratching.

6. *Care of the Base of the Ulcer.*—Where there is a clean granulating base, the graft can be applied directly upon it. In other instances, the base may be leather-like, due to repeated healings, scarrings, and breaking down of the tissue. In these cases, it is best to excise this base completely with the surrounding edge and apply the skin graft directly on the new site. With such excision the tissue under the scar herniates through the cut fascia, showing the tension under which the fascia and the underlying tissues had been placed by the previously scarred areas. Near the ankle such an incision may expose bone, but the graft will take better on this bone than it would on the old scarred ulcer base. The skin develops from two embryonic layers. The outer layer, the epidermis, is derived from the ectoderm and is attached by the dermis. The epidermis consists of stratified epithelium in four layers and varies in thickness. The corium or true skin is of mesodermal origin. It has no direct blood supply and depends upon the dermis for its life. The skin should be placed upon living tissue

* Bishoff

Secondary tissues consist of granulations in which are capillaries and fibroblasts The end result of such tissue is scar Skin grafts placed on such tissue do not take satisfactorily It is fundamental therefore that the base be a healthy vascular one which is bathed in tissue fluid

7 *Preoperative Antisepsis* —Harsh antiseptics interfere with graft takes In the local preparation of the site the only solution used by our Clinic is a sulfonated detergent soap containing Hexachlorophene (pHisoHex) No dye or antiseptic is used

8 *Ambulatory Preparation for Grafting* —Where the patient must be ambulatory during this preparation a modified Unna type of boot or bandage will aid in the preparation This causes pressure at the site and constricts the veins which in the venous type of ulcer will aid healing in most patients The antibiotic therapy and muscle adenylic acid described above can be given at the same time

9 *Streptokinase and Streptodornase (Trypsin)* —Certain biologic substances have been found to have an autolytic effect on necrotic tissue These substances dissolve the eschars of burns the base of decubitus ulcers, necrotic bone and soft tissue abscesses some sinuses and fistulas, as well as hematomas In certain patients the necrotic base of an ulcer can be prepared in this way These substances have a predilection for dead and not viable tissue and liquefy the fibrous and necrotic debris They are formed by the action of streptococci and are known as streptokinase and streptodornase

This is a fibrolytic effect The activating agent is Trypsin A satisfactory dosage has been found to be 20 000 S K and 5 000 S D applied locally The effective action in the liquid form is one hour and in a gel form three hours.[11]

**Preoperative Preparation of the Donor Site** —The area from which the graft is to be taken should be selected for size and availability If there has been phlebitis the affected limb should not be used The part should be dry-shaved without abrasion the day before It is then surgically cleansed with the sulfonated detergent soap

Antiseptic solutions may destroy the epithelium

## ANALOGOUS GRAFTS

Types —1 **Electric Dermatome Grafts** —Where a large area is to be grafted, the motor driven dermatome introduced by Brown[3] will furnish an even adequate graft which can be uniformly cut. The dermatome is set for between [illegible]/1000 to [illegible]/1000 of an inch in thickness These grafts can be taken as uniform strips 3 to 4 inches in width. The exact size desired can be measured and cut, and this careful fit contributes to the high percentage of takes of the graft. This type of machine has revolutionized grafting and can be used successfully by even the inexperienced The machine appears to take the grafts smoothly and without trauma to them. A new blade must be used each time. The thin grafts take the best. The thicker grafts stand the stress of use the best.

The graft may be sutured with fine steel wire No 36 or No 37 or five-0 silk attached to a minute eye-type of needle Blood and plasma clot is a glue and will aid in attaching the graft.

**Attachment of Graft.**—(*a*) *Open Method*—The graft may be attached and left open This method can be utilized only where the area is not subject to trauma or other disturbance This eliminates the use of this method on children A minimal amount of suturing, adequate approximation of the graft in size and shape, and the utilization of the patient's own clotting factor as glue aids in the take

*b* *Dressings*—Pressure padding has been the time-honored method in holding grafts in place If the wound is clean, pressure provides good graft takes. If the recipient site is not surgically clean, these are odorous, often secondarily infected, and sometimes painful If infected, these can be kept wet with a penicillin-saline solution

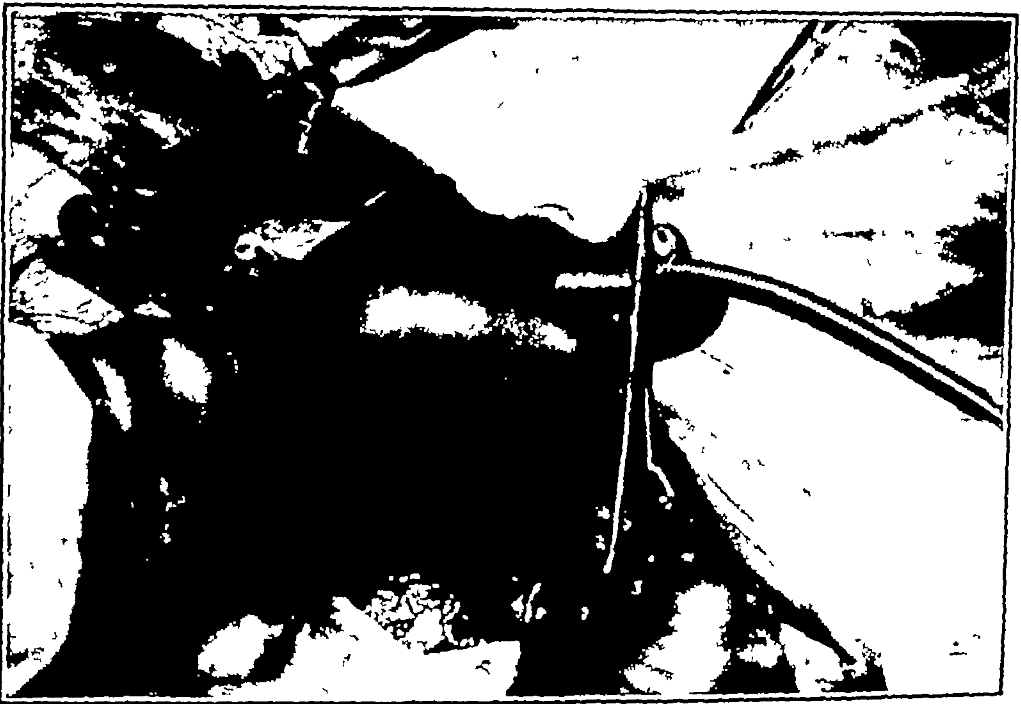


FIG. 243—Electric skin grafting machine (Brown) removing section of skin Piece of skin being lifted is shown to right of picture (Pratt, courtesy J A M A)

*c* *Backed Grafts*—Grafts may be backed with bobbinets These can be anchored with a rubber cement or adhesive tape The bobbinet may be made of cotton, nylon, rayon or plastic, and its use reduces the amount of suturing required

*d* *The Use of Adrenal Cortex*—ACTH has helped in the healing of burns and has a place in some grafting Its direct action has been thought to be the control of fibroblastic proliferation Its beneficial effect may be due to increasing the metabolic activity of normal cells which are depressed in the wound [8]

2. **Other Dermatome Grafts.**—The use of such apparatus as the early Padgett or other nonelectric dermatomes has been replaced in most clinics where the motor driven machine has been tried These dermatomes have been of great value in the past in raising relatively large grafts, but their use today is restricted. The more rapid work of the electric machine makes

it the preferable one When it is not available the type of dermatome which depends upon cementing of the skin to the drum has its place This machine requires skill and experience and an extremely sharp blade which has not been injured in its handling These drum methods are preferred to



FIG 244 —Pieces of donor skin removed and being stitched together for replacement on muscle in lymphedema operation

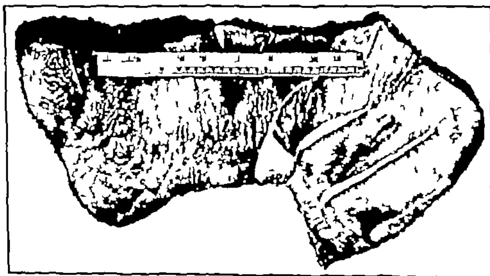


FIG 245 —Tissue removed at lymphedema operation Note the epithelial layer has been removed prior to tissue excision

the older Thiersch or pinch grafts Other electric dermatomes are now available (Padgett, etc )

3 **Thiersch Grafts.**—Where a motor dermatome is not available grafts can be raised and placed by one of the older methods that of the Thiersch type being satisfactory These grafts can be raised with a sharp razor

a skin graft knife or an autopsy knife. This knife must be "hair" sharp, and it should be sharpened after each use The skin is held on tension with wooden blocks, one held by the operator and one held by the assistant, so that a flat skin surface is presented The knife or razor is then held in a horizontal fashion parallel to the skin The graft is raised by a sawing back-and-forth motion of the blade on the flattened skin surface With some experience, a somewhat uniform-sized graft can be raised, if the knife is in excellent condition, a very large graft can be prepared in this way The graft will accordion itself up on the knife blade

This graft then may be tacked in place with fine silk or wire Sutures may not be necessary If the part is bandaged care is taken to prevent slipping of the graft

4 **Pinch Grafts.**—Pinch grafts usually are obtained from the thigh or abdomen The same care in preparation of both the donor and recipient sites is taken Excess granulations on the recipient site should be removed These can be cut away with a razor blade, with a scalpel, or with a curette

Pressure with a saline dressing usually will stop the oozing while the graft is being prepared If the part is inflamed silver nitrate can be applied Normal saline is then used to wash off the silver nitrate and slough

A straight Kieth needle point is inserted into the skin and a small segment of skin is lifted up This can be excised with a razor blade or with a sharp scalpel The graft is placed directly on the donor site from the needle point Each graft is approximately $\frac{1}{4}$ inch in diameter They should not be placed beneath the subcutaneous layers

Each of these skin graft points then becomes a germinal center for the proliferation of epithelium, and this proliferation usually occurs in all directions from the graft These grafts will take at times when other grafts fail The wound is dressed carefully and pressure applied. Scarring is to be expected in such grafts.

5 **Full Thickness Grafts.**—Full thickness grafts are of use in some vascular ulcers They are particularly of value where a large defect exists which exposes the tendons In the luetic type of ulcers these grafts will take well after the underlying disease has been treated Where a vascular ulcer has developed secondary to trauma, with loss of tissue, a full thickness graft is also effective

There must have been adequate skin preparation both at the recipient and donor sites The grafts must be cut in an exact pattern A simple way to prepare such a pattern is to place a gauze sponge on the ulcer site Serum or blood will make an exact reproduction of this ulcer, and this, in turn, can be pressed on the donor site to outline the graft which must be raised This pattern also can be cut from sterile cellophane Cellophane can be placed over the ulcer and the outline of the ulcer cut exactly This pattern then can be outlined with a scalpel point on the donor area

A sharp scalpel is used to raise the graft Fine small forceps can lift the area as it is raised, and the full thickness graft is removed slowly and carefully without any attached subcutaneous tissue or fat The graft is then transferred to the recipient site and tacked in place, with the fine wire sutures mentioned on page 743 tied very loosely If the area is dressed, uniform pressure must be applied and maintained

The donor site of all ulcers can be treated locally with a pressure dressing of petrolatum gauze and the wound is not dressed for a period of ten to fourteen days

These full thickness grafts should not be used indiscriminately They have a definite place in the therapy of ulcers but this should be reserved only for those in which a full thickness graft is indicated The recipient site should not be disturbed In all cases our results have been better when these recipient sites were wetted through with sterile saline twice a day Penicillin may be added if there is infection

7 **Pedicle Grafts** —These grafts are used rarely in vascular disease but they do have a place (see Lymphedema p 701) If tendons or other deep tissues are exposed these grafts may help The pedicle graft must be raised in sections

**Postoperative Care of the Graft** —The graft must lie on the recipient surface in order to be successful. Some grafts are lost because the graft while accurately approximated on the edges was not in good contact with the bottom of the area to be grafted either due to its riding up or due to an intervening layer of blood Any collected blood should be released at the end of the operation

a. *Dressing* —A waxed mesh gauze is applied over the graft If a dressing is used it should be the exact size of the ulcer Accurate and even pressure is applied A slip or bump may displace the graft before it is settled in place The limb should be immobilized by either a wooden or wire immobilization splint in order to counteract a tendency to foot drop In some a cast is applied The grafts taken with the electrically driven dermatome heal best when left opened or covered with a dry dressing If dressed they are left alone for fourteen days to permit healing If one inspects such a graft too early the epithelium will be disturbed before it is adherent and some of the graft will be lost.

b *Wet Dressing* —If the graft appears to be infected or there is a mal odor the graft can be moistened with saline solution to which is added penicillin in strength of 300 000 units to 250 cc saline

The Thiersch and pedicle grafts seem to take better when they are kept moist with sterile saline The saline can be applied to wet the grafts twice a day The sterile saline solution freshens and moistens the graft and tends to take away any serum or blood collection The saline solution is applied with a dropper to avoid trauma

c *Antibiotic Therapy* —Where it has been difficult to sterilize the site of the graft or the ulcer has been present a long time penicillin solution (300 000 units to 250 cc saline) may be applied on the graft postoperatively and this has worked well. In such cases general penicillin or another antibiotic also is given

d *Re-dressing* —The first dressing should be performed carefully after moistening the graft thoroughly each layer of gauze being teased away separately The last layer need not be removed until it falls away by itself

After the dressings are removed the graft is exposed. This exposure hardens the graft so that it can withstand the pressure of stocking and shoes Applications of potassium permanganate in extremely weak solution (1 25 000 or 1 35 000) will help reduce the fungus pr[illegible] Sterile saline soaks may be used Whirlpool baths also help

Care is taken to prevent injury to the graft for several weeks In certain cases, weight bearing is delayed in order to avoid disturbing the graft

**Postoperative Care of the Donor Site.**—The donor site is dressed with petrolatum gauze. At times, where there is any question of inflammation, antibiotics are used This donor site is not dressed for a period of ten days to two weeks At that time a firm, healed area usually will be present When it is disturbed too early, granulations and at times secondary inflammation develop The healing may be long delayed and disfiguring scars may result

**Recovery of Sensation in Grafted Areas** —There has been a divergence of opinion as to the recovery of sensation after skin grafting In pedicle grafting under favorable conditions, sensation begins at the region of nerve supply and spreads peripherally Pain sensation first appears in three months and touch is felt in nine to twelve months The sympathetic innervation recovers likewise After split-thickness grafts, sensation usually is patchy and incomplete according to most observers[3,6,10,15] McCarroll[13] reported that there was complete recovery of sensation depending on the thickness of the graft. Our own experience in the massive grafting performed in the lymphedema operations (see page 743) showed that there was recovery within six months in those that did not form keloids. This return was the same with pain and touch although the latter returned later (one year).[16,17] Our patients had their superficial nerves often completely destroyed but apparently developed new ones This observation is in contrast to the work of Kernwein,[9] who considered it necessary to have subcutaneous fat and sensory nerves intact for sensation In our patients no fat and little nerve tissue is left

## HOMOLOGOUS GRAFTS

The severe injuries encountered in war, burns, atomic explosions and air or automobile accidents at times make the supplying of skin to the injured patient impossible It has been known for many years that homologous grafts, except in the case of identical twins, would take well but later would melt away and be destroyed by the host, as it destroys every foreign substance including blood which is introduced into it That this destruction took time was known, and this time factor has been utilized as a lifesaving measure With the possibility of an atomic war in view, the *wide use of homografts of skin is under study* The enormous amount of skin that would be needed can hardly be realized It is known that the most severe injuries from an atomic explosion except at the site of the explosion will be burns of a thermal nature Since 80 per cent of those that survive such an explosion will have surgical burns, the possibility of utilizing this method for their immediate therapy is considered The survival of such skin grafts for a variable time is accepted and some observers believe that all the skin is not lost[1,12,14,20]

There are three theories as to why the host destroys the grafts These are (*a*) immunity due to blood incompatibility; (*b*) differentials in the cells causing a reaction around the cell and its destruction, (*c*) the protein-

of the grafted tissue are antigenic and an active immunity to them develops. This latter theory seems most tenable and has been proven to some extent by Baxter.[1] Subsequent grafts from the same donor last shorter periods of time.

## SKIN BANKS

In our work on lymphedema we have kept autografts three and four weeks at ice box temperature (4° C.) and used them satisfactorily. It is our belief that homografts can be preserved for a like time and used satisfactorily although the host will destroy them eventually. This method of grafting is important in severe burns and would be valuable for warfare or atomic burns.

In our preservation of skin, specimens were placed in a sterile boat to which 20 cc. of normal saline were added. Three hundred thousand units of penicillin were in the saline solution. This boat was placed in a sterile rubber glove and was closed with rubber bands. The specimen was placed in an ordinary ice box with 4° C. temperature.

The preservation of homologous grafts has been studied at the Tissues Bank of the Naval Medical School at Bethesda, Maryland. The skin is removed with a dermatome and wrapped around a glass cylinder with the inside surface out. This skin is held in place with perforated cellophane. The skin is then placed in a Ball type fruit jar. The preservation medium consists of Earle's buffered salt solution (90 per cent), pooled human plasma (10 per cent) and 50 units of penicillin and streptomycin per 1 cc. A phenol red indicator is added. As the acid products of metabolism accumulate, the indicator will change color (at pH 7). The solution is changed when the color change occurs. These grafts have taken well after storage as long as eighty-four days.[7,22]

At the present time there is no substitute for fresh autogenous skin grafts. These grafts can be kept up to perhaps one hundred days. The future of skin homografting depends upon our ability to alter the antigenicity of the donor tissue. So far this has not been accomplished.[22] Attention to blood typing may be important.

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# SECTION VIII

## Special Cardiovascular Technics

## Chapter 41

# SURGICAL TREATMENT OF PORTAL HYPERTENSION AND ESOPHAGEAL VARICES

*Emergency Treatment of Bleeding, Shunts, Ligations, Splenectomy, Esophagogastrectomy, Resection of the Varices, Injection of Veins in Esophagus*

THE primary interest of the medical profession is to prevent death. The secondary function of the physician or surgeon is to relieve pain and then to treat the lesion or disease which is endangering the individual or causing the pain. To fulfill these fundamental requisites, often it is necessary to treat a symptom or complication rather than to attack the underlying problem. The surgical treatment of esophageal varices is such an example. The varices are secondary to a condition of portal hypertension which follows some type of block in the portal system. The immediate danger is in the varices which are secondary to portal hypertension. The patient's life is in danger due to hemorrhage from these varices. Thus, the surgical problem is to control this bleeding. The problem of treating the underlying cause, once the liver changes are advanced, still eludes us. It has been demonstrated, however, that life is compatible with advanced liver pathology and even with portal system obstruction provided the hemorrhagic manifestations of the disease can be rectified. The treatment of cirrhosis of the liver, despite surgical adventure, is a medical problem. Its success depends upon dietary control, vitamin supply and the elimination of toxic factors. It is only in the complications that the surgeon can assist. The small amount of liver required for life and function is astounding.

Portal hypertension may be caused by primary cirrhosis of the liver or secondary to hepatitis, Banti's disease, congestive splenomegaly (Rousselot)[20] and schistosomiasis of the liver. Most such conditions result in esophageal varices which are thin walled, may rupture and cause hematemesis. These esophageal varices result eventually in fatal hemorrhage.

The evolution of the present concept of the therapy of esophageal varices has been by trial and error. Banti's original concept that the disease began primarily in the spleen and the liver changes were secondary probably was erroneous. The German pathologists' views that portal bed block was more rational was emphasized in 1936 by Rousselot.[77] This work led to the term "portal hypertension." The possibility of using some type of shunt such as the Eck[14] fistula to reduce the excess tension in the portal system was first suggested by Thompson, a hematologist in the Spleen Clinic at the Presbyterian Hospital, New York City, in the 1930's.[77]

The first operation of this type was performed in 1942 by Rousselot, assisted by Whipple This operation successfully united the ileocolic vein to the vena cava, and the patient is alive today[27 30a] It became evident that larger veins must be anastomosed if thrombosis was to be avoided Tube anastomosis thereafter was tried by Blakemore[4] and the use of sutures by Whipple[38 39] Blalock,[7] Linton,[18] Rousselot[28] and others standardized the technic.

The patient with esophageal varices is a potential bleeder Like a hand grenade or dynamite, he may "blow up" at any moment Fifty to 80 per cent of the patients will die of hemorrhage within one year and a half of their first bleeding accident The tension in the portal system gradually increases, and the secondary varices in the esophagus and stomach enlarge and rupture readily with massive hemorrhage

**Anatomy.**—The portal vein system receives the blood from the entire digestive system, the pancreas, the gall bladder and the spleen This blood is filtrated through the liver sinusoids or capillaries and then enters the systemic vena cava through the hepatic veins The system has no valves and is unique in that its blood passes through the capillaries of the organs it drains and then through the capillaries of the liver The number of variations from the normal are recognized now with the anatomical studies by Douglass *et al*,[12] Michaels,[21] and Madden [19]

There may be an extra- or intrahepatic obstruction to the system Blockage of this system thus may occur outside or inside the liver

The portal vein is approximately 4 inches in length It originates at the level of the second lumbar vertebræ by the junction of the superior mesenteric and the splenic veins These veins join together to form the portal vein in front of the vena cava and behind the neck of the pancreas

The portal vein passes up in the right border of the lesser omentum behind the upper part of the duodenum to the porta hepatis, where it divides into a right and left branch and enters the liver with the branches of the hepatic artery In the lesser omentum, the portal vein is behind and between the common bile duct and the hepatic artery It is accompanied by lymphatic vessels, glands, and nerves

The branches of the portal vein are the splenic, the superior mesentery, the coronary, the pyloric, the cystic, and the parumbilical veins. These lesser branches receive tributaries such as the gastric, the pancreatic, the inferior mesenteric, the colic, the intestinals, the sigmoid, and the esophageal branch veins

With portal vein obliteration, these branches of the portal vein secondarily dilate, particularly those in the esophagus (through the coronary vein) Other enlargements of the portal vein occur in the stomach, the colon, the duodenum, and the left renal vein.

The coronary veins drain both sides of the stomach, running from right to left along the lesser curvature At the esophageal junction these veins receive blood from the esophageal veins These veins then reverse themselves from left to right to empty into the portal vein In addition the azygos and hemiazygos veins run on either side of the esophagus These veins anastomose with branches of the coronary and splenic veins and thus create some communication between the portal and caval systems These

veins are in the submucosa and have poor support. With a block in the portal system the back pressure quickly dilates the coronary and esophageal veins and varicosities develop. Gray[16] called attention to the similarity of the azygos and the hemiazygos to the greater and lesser saphenous veins in the development of pathologic varicose veins. That these veins may be a factor in some hemorrhage may be accepted.

The knowledge and conception of the normal liver is increasing but is far from complete. The liver has a great functional reserve in its vascular system. The circulation in a lobule or in many lobules may be inactive, partially inactive or fully active at varying times. At any one time, 75 per cent of the liver is inactive unless it is excited. Thus there seems to be a storage phase when the liver sinusoids are full of motionless blood cells and a nonstorage time when the sinusoids are practically empty of blood cells. The intercommunications of the arterial and portal systems also are under study. According to Wakim and Mann[37] these are:

1. Interconnections at the presinusoidal level of the arterial and portal branches in the intralobular spaces.
   - (a) Side to side direct anastomosis between the artery and vein.
   - (b) Side to side connections between the artery and vein, the vessels continuing their independent course to the sinusoids.
   - (c) Arterioles terminating end to side into the portal venules.
2. Interconnections at the sinusoidal level.
   - (a) The terminal branch of a hepatic artery and a portal vein opening into the same sinusoid.
   - (b) Cross communications between the arterial and portal sinusoids. The presence of various sinusoids has been demonstrated. These are:
     1. Arterial sinusoids. The vessel is a terminal branch of the hepatic artery.
     2. Portal sinusoids. The vessel is a terminal branch of the portal vein.
     3. Arterial and portal sinusoids. The mixed portal and arterial blood results from terminal branches of the arterial and portal system entering the same sinusoids or cross communications between the arterial and portal sinusoids. The vein of exit from such sinusoids is a central or hepatic vein.

Banti's original conception that portal hypertension began in the spleen and was followed secondarily by cirrhosis of the liver delayed acceptance of our knowledge that patients may have portal hypertension with a normal liver. Thus we have extra- and intrahepatic blocks. In the former the block of the portal vein is outside the liver. In the latter the portal obstruction is within the liver and is usually due to cirrhosis. Portal pressure assists in the determination of the site of the block.

(1) The *portal vein system* is a large system of branches without anastomosis going through five or more divisions to the sinusoidal circulation. The branches are at right angles to the main vein and the sinusoids arise at the venule tips.

(2) The *arterial system* is close to the portal vein and may wind around it The branches of this system (hepatic artery) are of three types (*a*) vaginal branches—which form an arteriolar plexus in the portal spaces, supply its blood and end in capillaries which communicate both with the portal vein and the intercellular sinusoids, (*b*) vascular branches, which end in the sinusoids at the outer edge of the portal space, (*c*) capsular branches, which form an arteriolar anastomosis and communicate with the internal mammary, phrenic, the renal and suprarenal arteries

(3) The *hepatic venous system* commences with the central veins, divide into the sublobular veins and eventually reaches the inferior vena cava These veins receive their blood from the tributary sinusoids throughout their length This system is unlike the portal veins, which receive their sinusoids only from their tips [20] The two venous systems are separate and the hepatic one does not connect with the portal vein or the hepatic artery except through the sinusoidal bed

The portal vein supplies 60 per cent of the hepatic blood flow, the remaining 40 per cent coming from the hepatic artery This balance is maintained by the splanchnic nerves This regulates the flow from the high pressure artery to the low pressure portal system to a common level at the sinusoidal site, and guarantees oxygen supply if some pathology interferes with one or the other of these circulations Occluding the portal vein raises the hepatic artery flow from 50 to 100 per cent [35a]

## CIRCULATION OF THE LIVER IN CIRRHOSIS

The liver cells, which obtain their blood supply from the intervening sinusoids, are deprived of blood supply as the intercellular sinusoidal spaces are compressed Thus, the early obstruction is at the sinusoidal level The interconnections between the arterial and portal systems at the presinusoidal level therefore become important The portal and then the arterial blood is diverted The portal pressure rises 1 mm of Hg for every 40 mm of arterial pressure in the normal liver In the cirrhotic liver however, the rise is 1 mm of Hg for every 6 mm rise of arterial pressure.[17] Thus we have an active arteriovenous perfusion caused by the resistance of blood flow through the sinusoids This acts somewhat like an arteriovenous aneurysm and shunts arterial blood away Forty to 45 per cent of the arterial blood may by-pass the liver cells by way of the presinusoidal arteriovenous communications [20] As a result of cirrhosis, the total liver vascular bed is reduced, the portal veins are distorted, and the hepatic veins, likewise, are affected With destruction of the liver cells, the central veins are pushed to the periphery of the lobule and bound in scar The hepatic cells become isolated instead of forming a continuous sheet between the portal and hepatic venous trees Whole liver lobules may be scarred and side-tracked from the portal blood supply The arteries persist, however In such cases, the blood supply to the parenchyma for liver metabolism is dependent upon the hepatic artery The patient dies only when this latter mechanism fails A very small supply of hepatic tissue with normal blood supply is necessary for liver metabolism (much less than is present in the most advanced cirrhosis) Therefore, a shunt diverting some of the

portal stream from the liver may permit the arterial supply to nourish the liver parenchyma better

The intrahepatic blocks most often are due to a cirrhosis of the liver The cause for the cirrhosis is not clear but probably it is due to metabolic disturbances An avitaminosis from faulty or irregular diet seems a clear cause Alcohol *per se* probably is not a cause except as it indirectly affects the patient's habits and diet Poisons and other toxins which inflame the liver as well as inflammatory or infectious attacks on this organ may result in the fibrous tissue deposit and scarring which secondarily leads to cirrhosis Those interested in the medical aspects of cirrhosis are directed to the many and complete treatises on this subject

The extrahepatic blocks may be due to any pressure on the portal system or its contributory veins Thus tumors thrombosis congenital anomalies trauma to the spleen or its vessels and cavernomatous transformations all may be factors in the development of extrahepatic portal obstruction

**Symptoms** —As the result of portal obstruction certain definite symptoms develop Of these a congestive splenomegaly occurs early There is a secondary anemia which is marked and may be disabling With this is a thrombocytopenia and a leukopenia There may be all of the signs of secondary vein obstructions in this splenic area the stomach small and large bowel the esophagus pancreas and the gall bladder

Two main symptoms are of surgical importance Due to the blockage there may be *ascites* with collections of fluid in the peritoneum and other serous cavities As a result there is a back pressure on the entire venous system with edema and anasarca Twenty five per cent of those coming to operation have ascites Most of the ones with ascites can be relieved with medical management The second and more important symptom of surgical importance in this group is esophageal varices with *hemorrhage* The incidence of bleeding is 33 per cent. Of those who bleed, 50 per cent die within one year [illegible] It is for this latter symptom that surgical intervention has been tried and to a great extent has been successful.

Child[illegible] showed that the *Macaca Mulatta* monkey will tolerate a sudden and complete occlusion of the portal vein The efficacy of the collateral circulation in undiseased animals thus is proven The variations in the slower occlusions which occur in the cirrhotic patients must differ from that of slow occlusion in the systemic vessels [illegible]

**Treatment of Esophageal Varices** —To treat this problem of hemorrhage from these dilated veins in the esophagus and upper stomach is physically difficult. Their site makes exposure a problem their constant irritation by the passage of food and drink increases the incidence of rupture and the pathologic increase in the blockage in the portal system as the disease progresses makes certain that the vein tension will continue to rise

**Emergency Control of Hematemesis** —The possibility of controlling sudden hemorrhage in the intestinal tract by that oldest expedient pressure has been considered and tried in many ways. A latex bag attached to the end of a Miller Abbott tube or some other tube placed in the esophagus and inflated was presented by Rowntree and his co-workers [illegible] This is an excellent way to control the sudden bleeding and can be prepared quickly when needed It has been left *in situ* as long as four days The addition

of a feeding tube to extend beyond the tamponade point makes it possible for the feeding requirements to be maintained despite the tamponade This method has been improved by a gastric and an esophageal tube for compression of the varices The two balloons retain the compression in the varices area. These tubes are radiopaque so that one can determine their site [32]

## OPERATIVE THERAPY

The successful anastomosis of the portal system to the systemic circulation empties portal blood into the general circulation and reduces the portal pressure

**Roentgen Ray Visualization of the Spleen and Portal System by Splenic and Portal Portagrams.**—The importance of the knowledge about the circulation in the spleen prior to operation is apparent Direct injection of dye into the spleen has supplied this information The spleen can be successfully injected prior to operation For technic see Chapter 39 on Roentgen Ray Visualization, pages 724 to 740 When the abdomen is opened, the injection of dye into the mesenteric vein gives *x*-ray visualization of the portal vein system Both of these technics have been successfully established by Rousselot, Ruzicka and Doehner [11a,30a] (See pages 734, 735 to 738 )

The technical difficulties of such operations are tremendous As a result of the portal hypertension, the vascularity of the entire area is greatly increased Many surgeons have discontinued the operation before opening the abdomen because of the enormous number of vessels encountered and the blood loss The meticulous asepsis necessary is also a difficult problem to surmount, as the slightest infection in cases of this kind will produce a thrombosis even when a perfect anastomosis has been achieved Efforts to replace suture methods with tubes were unsuccessful The anastomoses

anastomosis The renal fat is then opened the left kidney delivered and elevated. A Potts-type clamp is placed on the renal vein The splenic vein is then anastomosed in an end to side fashion to the renal vein at the point protected by the Potts clamp The addition of the femoral vein graft to this technic materially shortens the operation time and the danger of tearing the splenic vein In Rousselot s series of nearly 50 patients successful anastomoses have been completed in 80 per cent Eighteen of these have been done with femoral vein grafts The Potts clamp is released and then the seraphin clamp is removed from the splenic vein It is necessary that the splenic vein be anastomosed with due regard to the femoral vein s

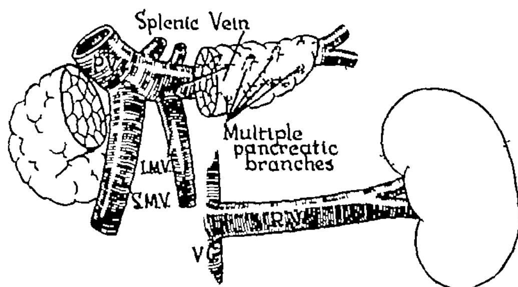



FIG 240 —Splenectomy and splenorenal shunt by vein graft (Rousselot)

valves The technic of anastomosis in vein grafts is described on pages 340 to 341 A simple running suture of an over and over type has proved satisfactory The wound is closed with careful hemostasis

2 *Technic of Portacaval Operation* —Under careful aseptic conditions the abdomen is opened through a right rectus or pericostal incision All vessels are carefully ligated

The portal vein is isolated, mobilized and dissected free. It is sectioned near the liver The liver end of it is transfixed and ligated The portal vein is then anastomosed to the vena cava by an end to side anastomosis. The vena cava must be mobilized for a distance of four inches The end of the portal vein is anastomosed to the side of the vena cava in a side to end manner similar to that described under splenorenal anastomosis.

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(1) an end to side anastomosis of the splenic vein to the left renal vein, (2) a shunt from the portal vein to the vena cava, (3) splenectomy (this splenectomy may be combined with splenorenal shunt), (4) ligation of the splenic artery, (5) ligation of the hepatic artery; (6) esophagogastrectomy, (7) a combined splenectomy and devascularization of the esophagus and stomach, (8) mediastinotomy and packing, (9) obliteration of the coronary veins by suture, (10) the injections of the varices through an esophagoscope

1 *Splenorenal Shunt by Autogenous Vein Graft with Splenectomy* (Rousselot).[27]—Through a left thoraco-abdominal incision the abdomen is opened The viscera is packed off and the liver and spleen inspected The spleen is freed and elevated and the branches interfering with the mobilization of the splenic vein are ligated with fine silk. This is tedious and difficult The branches to the pancreas particularly interfere with this mobilization and the tension in such vessels makes their control hazardous The spleen is removed To facilitate this operation and to decrease the bleeding and the danger, Rousselot uses a section of the femoral vein to make the shunt from the splenic to the renal vein This decreases the amount of dissection necessary to get sufficient length of splenic vein for a direct

anastomosis The renal fat is then opened the left kidney delivered and elevated A Potts-type clamp is placed on the renal vein The splenic vein is then anastomosed in an end to side fashion to the renal vein at the point protected by the Potts clamp The addition of the femoral vein graft to this technic materially shortens the operation time and the danger of tearing the splenic vein In Rousselot s series of nearly 50 patients successful anastomoses have been completed in 80 per cent Eighteen of these have been done with femoral vein grafts The Potts clamp is released and then the seraphin clamp is removed from the splenic vein It is necessary that the splenic vein be anastomosed with due regard to the femoral vein s

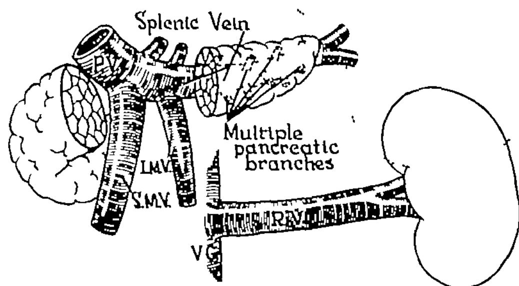



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This can be done by partly closing the vena cava with a Potts-type clamp or by other clamps If regular clamps are used, the clamps are then released in the following order first the distal one on the vena cava, then the one on the portal vein, and finally, the proximal one on the vena cava

Care must be taken to avoid angulation of either one of these vessels, particularly at the time of the replacement of the viscera The mortality in the hands of the general surgeon will be high until the technic is mastered.

It is to be hoped that a better technic for handling this problem than either of these operations may be evolved These operations are difficult, time-consuming, technically dangerous and at times impossible to complete

3 *Splenectomy* —Splenectomy alone has not controlled satisfactorily the bleeding from esophageal varices Whipple mentions "the discouraging follow-up problems in the recurring hemorrhage in patients who have had a splenectomy for congestive splenomegaly or Banti's syndrome "[39] Splenectomy in addition to a shunt procedure, however, is helpful Other vessels have been used for shunts The mesenteric and other systemic veins have been anastomosed The femoral vein *per se* may prove to be the ideal one.

4 *Ligation of the Splenic Artery* —Blain[6] suggested ligation of the splenic artery in 1918 for Banti's syndrome One of his patients lived thirty-four years [6] Everson and Cole[15] have revived the procedure in the poor risk patient It has been used in extremely poor risks but the results have not appeared to justify its routine employment

5 *Ligation of the Hepatic Artery* —The fact that in Banti's disease changes occur in the liver which make the ligation of the hepatic artery possible has been proven In some autopsy specimens there was complete occlusion of the hepatic artery It is apparent that in such patients arteriovenous fistulas develop in the liver with sufficient collateral to make the hepatic artery supply unnecessary Reinhoff[26] has advocated the ligation of the hepatic, splenic and left gastric arteries in the treatment of intrahepatic portal hypertension His results reportedly have been good The anatomic or physiologic reason for this procedure has not been substantiated The effect of ligating the artery has been proven detrimental and the procedure must be considered not acceptable at this time [19]

6 *Esophagogastrectomy* —Phemister and Humphreys introduced a direct attack on the varices with the resection of the esophagus and part of the stomach They reported some good results This is a formidable procedure because of the enlarged veins [25]

7 *Devascularization, Splenectomy and Vagotomy* —More recently Gray[16] has enlarged on this operation by devascularizing the lower part of the esophagus and the upper part of the stomach, performing a splenectomy and doing a bilateral vagotomy The reason for the latter procedure is to abolish the cephalic phase of gastric secretion With the vagotomy it is necessary to perform a gastroenterostomy This extensive surgery on such poor risk patients has not been accepted by most of the profession

8 *Mediastinotomy and Packing* —Packing the posterior mediastinum with gauze as an aid in developing a new collateral circulation and shrinking the veins in the esophagus has been reported [34] The rationale for this effect is not apparent

FIG 247 —Preparation of autogenous femoral vein graft in upper picture Lower picture shows end to side anastomosis of the splenic to the renal vein using the graft.

9 *Obliteration of the Coronary Veins* —Many surgeons have felt that the decrease in the portal hypertension was an indirect attack on the problem of bleeding esophageal varices Several have tried a more direct attack The obliteration of the veins themselves seems a logical step. Crile has tried such an operation by suturing the redundant mucosa with a running suture placed below the varices This procedure has worked well in those patients with extrahepatic blocks [11]

10 *Injection of the Varices Through an Esophagoscope* —This treatment was first tried by Frenckner and Crafoord[10] of Stockholm in 1939. Moersch[22] and Patterson and Rouse[24] have reported successful results since that time The vessels are visualized and injected with five per cent sodium morrhuate as often as needed Patients have ceased bleeding after this therapy It should be considered in all such patients and may have more application than it has been given so far

In summary, the increase in the venous pressure of the portal system due to either intra- or extrahepatic blocks produces a serious syndrome characterized by ascites and/or hemorrhage The latter complication causes the majority of deaths The corrective therapy has not been established. Ameliorization of the symptoms can be obtained by reducing the portal tension through some shunt to the systemic circulation A direct attack on the problem similar to that of enlarged veins elsewhere in the body, with some decompression procedure, would seem to offer the best hope therapeutically for the future.

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Chapter

# 42

# CAROTID SINUS SYNDROME; CAROTID BODY TUMORS

## CAROTID SINUS SYNDROME

BY CAROTID sinus syndrome is meant the signs of collapse which follow compression of the carotid artery at its bifurcations In patients who have this sensitivity, pressure on or near the carotid sinus will cause bradycardia, unconsciousness, and sometimes convulsions Hering[15] demonstrated that these symptoms were not due to pressure on the vagus nerve, a theory which had been accepted until 1927 In those without hypersensitivity, there may be untoward but less serious effects from pressure on this sinus

**Etiology.**—The reason the carotid sinus becomes hyperactive is unknown Certain knowledge about those who are afflicted, however, has been tabulated

1 *Predisposing Factors* —*Sex* —Males predominate among those afflicted by this syndrome The percentage is from 85 to 94 per cent [4,7]

*Age* —Most of the patients are in the fifty to seventy age group It is known that patients develop a greater tendency to become hyperreactors as they increase in age [25]

*Drugs* —Certain drugs accentuate the sensitivity Of these, the vagal type have the greatest effect Morphine, the nitrites, calcium, nicotine, thyroid, eserine, mecholyl, and chloroform all induce more sensitivity This untoward reaction to chloroform may explain the unexpected sudden deaths in the use of chloroform when it was a frequent anesthesia induction agent [3] Alcohol, on the other hand, has been reported as an inhibitor of carotid body activity [4 9]

*Family History* —The family tendency of inherent hyperreaction is shown by the fact that many patients have relatives with the same trouble

*Cardiac Activity* —The reflex varies with the cardiac rate It is absent with severe bradycardia, greatest with moderate bradycardia, and rare in those with tachycardia [29] Heart disease increases the incidence of the syndrome Ninety-three per cent of Sigler's patients with coronary sclerosis developed bradycardia on carotid sinus pressure [30] Thirteen of Evans' 20 patients had heart disease [7] Thirty-four of Nathanson's 40 patients were similarly affected [24] Cerebral arteriosclerosis appears to increase the sensitivity reaction [31]

*Emotional Upsets* —Most of the attacks occur in patients under emotional strain or fatigue This is particularly so in the cardio-inhibitory or vagal form The cerebral effect of the vascular response has been demonstrated [7–9 34 35]

*Afferent Stimuli from Other Parts*—Patients with biliary tract disease seem to be more susceptible to the syndrome[4]

2 *Precipitating Factors*—Sudden movements of the head or body may induce an attack. Sudden rising coughing shaving or the tight collar have been noted as incitors One of my patients developed syncope each time the barber pushed his head far to one side The incidence of the seizures is unknown but it is quite high in elderly males especially those with coronary disease

**Symptoms**—Three separate types of carotid sinus syndrome are recognized

A The *vagal or cardio-inhibitory* type occurs with a cardiac slow-down or asystole. The latter is preventable by the use of atropine One-third of the patients are in this group

B In the *vasodepressor* type the blood pressure drops This pressure fall is not related to the bradycardia One in 20 of those with the lesion is in this group and is unaffected by atropine This type of reaction can be prevented by the use of epinephrine

C The *cerebral* type is characterized by unconsciousness The heart is not slowed nor is there a drop in blood pressure Six out of every 10 with carotid sinus syndrome are in this category[9]

D There may be *mixed* forms[7,10] in which one or more of the above types of symptoms occur in the same patient

The carotid sinus syndrome is associated with syncope while the carotid sinus reflex is not The patients usually have an aura and some can prevent the activation of the syndrome during this period This syndrome is followed by dizziness weakness and epigastric distress Perspiration and pallor are prominent The attacks last from one to four minutes

**Diagnosis**—The history and symptoms are characteristic The diagnosis can be proven by inducing an attack Mild pressure only should be applied as heavy pressure and massage have been fatal[23,24] Pressure should be made on only one side of the neck. Hemiplegia has followed the application of such pressure[1] A differential diagnosis is necessary between this lesion and epilepsy Postdural hypotension may simulate the syndrome but is not reproducible by carotid pressure The vasovagal syncope or the more normal faintness with emotion fright the sight of blood etc can be differentiated by the history The heart disease patient who develops a heart block may cause difficulty in differentiation from this syndrome The syncope which occurs in aortic stenosis also may be confusing in the diagnosis

**Prognosis.**—Many patients have the sensitivity without spontaneous attacks In patients with severe signs there is a definite danger of fatal attack. Cerebral vascular accidents may be precipitated by the syndrome The prognosis in general depends upon the status of the underlying disease

**Treatment.**—*Medical Treatment*—In some patients no treatment is necessary other than having the patient understand the lesion and prevent action which precipitates attacks Sudden movements and the tight collar should be avoided Sedatives help ome patients and Banthine (beta-diethylaminoethylxanthene-9-carboxylate) may decrease the sensitivity of the sinus to the stimulus The use of the drugs atropine and epinephrine in specific types of reaction already has been mentioned

*Surgical Treatment.*—If the condition is severe an operation may be necessary. A procaine injection will determine what response may be expected from the operation. The sinus can be denervated. Periarterial sympathectomy has been most successful in alleviating the symptoms. Any pressure on the sinus, such as that caused by a tumor, is amenable to surgical therapy. *X*-ray therapy has been utilized with equivocal results.[33] The division of the glossopharyngeal nerve has been used where local operation has been impossible technically or unsuccessful.[26]

## CAROTID BODY TUMORS

**Anatomy.**—These carotid bodies are known as carotid glands or by the term *glomus caroticum*.[12] The carotid bodies are gray or red oval structures 5 mm. in diameter. They have a fibrous capsule which surrounds and subdivides them into lobes and lobules. They are situated at the bifurcation of the common carotid artery. Embryologically, they are of mesodermal origin, arising from the artery of the third branchial arch. They are composed of parts of the glossopharyngeal, vagus and sympathetic nerves in addition to the mesoderm tissue.[13,32] The nerves are distributed throughout the carotid body. Microscopically, the gland is lobular in shape. The carotid body cells, polygonal in shape with fine granular protoplasm, have an oval nucleus which contains chromatin. The cells are arranged in whorls which are contained by a supporting tissue in which there are many capillaries.[17,18] When these bodies develop into tumors the growth may wind around and involve the common, internal and external carotid arteries, the jugular vein, the vagus, the hypoglossal and glossopharyngeal and the sympathetic nerves. Sometimes these tumors encroach upon the pharynx itself.[14]

**Function.**—The normal function of these glands is not well understood. The early theory, that because they picked up chromium salts they produced an epinephrine substance, has been discounted. It has been suggested that they are chemoreceptors.[5,16,19,20,28]

**Etiology.**—The cause for the growth of the tumor is not clear. Most growths are so slow and benign, at least at the onset, that the average length of time this tumor has been present before therapy was sought was six years in Lahey's group.[20] The familial character of the lesion in some instances was shown by Lahey's reports of 3 siblings in 1 family who developed the tumor, while 2 others in the same family had been treated elsewhere for a similar lesion.

**Symptoms.**—Most of the tumors have mild or no symptoms. The symptoms develop as the tumor grows. They are usually unilateral, but bilateral growths have been reported. The tumors grow slowly. They are deeply placed beneath the sternocleidomastoid muscle. The mass is somewhat tense but compressible, and bruits develop in some. The tumor can be moved sideways but not vertically, since it is attached to the carotid vessels. In large growths the symptoms include signs following its projection into the pharynx and nerve pressure. Some patients have difficulty in swallowing. Some patients develop the signs and symptoms of carotid sinus syndrome. These patients require operative treatment.

**Diagnosis.**—The diagnosis is made on the history and symptoms of a slow-growing mass in the lateral part of the neck The lesion must be differentiated from the other lateral tumors of the neck

*Differential Diagnosis*—(a) *Branchial Cysts*—Such tumors are more superficial lower than the bifurcation of the carotid artery, and tend to bulge outward as they grow The carotid body tumors are deeper and project upward or inward rather than outward

(b) *Lateral Aberrant Thyroid Tumors*—If these tumors are multiple there is no difficulty in differentiation These latter tumors do not extend towards the mandible and can be moved up and down while the carotid body tumors are fixed in this direction

(c) *Metastatic Tumors*—A single carcinoma metastatic growth is unusual. Such an occurrence may require biopsy for differentiation The history growth elsewhere and the general symptoms of malignancy are of diagnostic aid

(d) *Neurofibromas*—These tumors can occur anywhere and if they are single at the site of the bifurcation of the carotid artery they cannot be differentiated without biopsy

**Pathology**—The pathologic picture of the carotid body tumors is confused because they have been described as adenomas neuroblastomas endotheliomas gangliomas etc The suggestion of LeCompte and Lahey[20,21] that we call them only carotid body tumors is realistic pathologically Grossly these tumors vary in size the largest being 190 grams[27] They maintain their lobulated shape These tumors usually are well encapsulated Some become so adherent to the artery that no line of cleavage can be developed The tumors are firm Microscopically they vary Most of them follow the normal structure with hyperplasia Another group has a cellular arrangement in which the cells are round contain cytoplasm and are placed in rows The chief cells appear epithelial in type The supporting tissue is restricted The third type is angiomatous in structure The cells are spindle shaped pressed together and have a massive capillary network to which they are opposed

In 15 to 50 per cent[11,13] malignant changes are alleged to occur Some investigators have reported that all of these tumors become malignant in time In such an event the cells change in size and shape invade the capsule and adjacent structures and have mytotic manifestations[21,22,24] LeCompte doubts that malignancy ever develops In none of Lahey s 22 patients was there conclusive evidence of malignant disease The recognition that malignancy is rare is important. In the past the idea that all of these tumors acquire carcinomatous tendencies has caused surgeons to remove them even when such excisions required ligation of the main blood supply to the brain This has resulted in hemiplegias perhaps unnecessarily

**Treatment.**—Where pressure causes obstruction or a carotid sinus syndrome develops excision is needed The diagnosis should be made before operation This is important in order to have discussed the dangers of complications should removal of the tumor be elected If it is likely that vessel ligation may be required carotid compression may be instituted for ten to fourteen days before operation This is a possible aid in reducing

hemiplegias, but not a certain one Three of Lahey's 7 patients who had major arteries ligated developed hemiplegia and died The author routinely obstructs the carotid artery with a rubber ligature for from forty-five to sixty minutes before dividing it if such division is necessary This has been successful as a prognostic point in all but 2 patients In one, where the vessel was ligated in continuity, a secondary hemiplegia was overcome by removing the ligature. In a second case, the hemiplegia did not develop until the second day, with a fatal termination This death proved at autopsy to be a cerebral hemorrhage In 2 patients reported by Conley[2] a hemiplegia was overcome by opening the ligated vessel and sucking out a blood clot with a catheter introduced into the cranium The introduction of a vein graft to by-pass the resected segment of artery is feasible In selected instances, an artery graft of an homologous type may be inserted See *Vein and Artery Grafts*, page 340

*Operative Treatment* —Many of these tumors may be excised without resection of the arteries This was possible in half of Lahey's[20] patients The danger of the freeing of an arteriosclerotic plaque in the carotid artery in an elderly patient exists and must be accepted if excision is elected

Operation is indicated in all these patients to confirm the diagnosis Biopsy should be done If the tumor is readily mobilizable, and can be removed without ligation of the vessel, this operation is advised Where the tumor is not large, where it definitely involves the common and internal or external carotid arteries, and where there are no pressure signs, excision may not be advisable

Where there are signs of pressure, encroachment of the vessels, carotid sinus syndrome or malignant tendencies, the tumor may be resected If the blood supply to the brain is interfered with, it may be re-established through a graft The possibility of needing a graft should be anticipated The jugular vein or the saphenous or femoral veins have sufficed as grafts

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# Chapter 43

# SURGICAL TREATMENT OF VASCULAR ULCERS

*Arterial Ulcers; Venous Ulcers, Specific Ulcers, Traumatic Ulcers; Ulcers Due to Blood Dyscrasia; Malignant Ulcers, Ulcers Due to Infection*

MOST vascular diseases appear inconsequential to the patient until complications arise The patient is disturbed by such complications as pain, interference with normal activity, and some lesion requiring extra care Vascular ulcers affect the individual in all three of these ways The physician is consulted only when this complication becomes disabling Ulcers may be due to an arterial deficiency of an organic or spastic type, chronic venous stasis, or specific lesions of an infectious, allergic, deficient or toxic nature They may also follow trauma, a blood dyscrasia or the changes secondary to malignant disease

A correct diagnosis is essential before attempting therapy This requires emphasis, for too often ulcers of different origin are classified together and all of them are treated with some type of boot, paste, or ointment It cannot be overstressed that the treatment is entirely different for each type of ulcer

## ULCERS OF LOWER EXTREMITIES

### *Classification*

I ULCERS DUE TO ARTERIAL LESIONS

A OCCLUSIVE ORIGIN

1 Arteriosclerosis or atherosclerosis with or without diabetes
2 *Thromboangiitis obliterans*
3 Embolic or thrombotic occlusion
4 Local pressure or trauma
5 Secondary to arterial aneurysm

B SPASTIC ORIGIN

1 Raynaud's syndrome
2 Scleroderma
3 Frostbite, pernio, immersion foot or trench foot
4 Local arteriospasm due to trauma or occupation
5 Glomus tumor
6 Reflex arteriospasm secondary to venous thrombosis

II ULCERS DUE TO VENOUS LESIONS

1 Varicose veins and their complications
2 Thrombosis and thrombitis
3 Congenital anomalies, hemangiomata and tumors

III ULCERS DUE TO ARTERIOVENOUS LESIONS
1 Arteriovenous fistulas and aneurysms
2 Arterial varices
3 Arteriovenous tumors

IV SPECIFIC ULCERS
1 Syphilitic
2 Tuberculous
3 Mycotic
4 Drug intolerance
5 Vitamin deficiency
6 Neurotrophic
7 Infection
8 Gout
9 Deficiency of muscle adenylic acid
10 X-ray radium and radioactive isotope exposure

V POST TRAUMATIC AND BURN ULCERS

VI BLOOD DYSCRASIA ULCERS
1 Polycythemia
2 Pernicious anemia
3 Leukemia

VII MALIGNANT ULCERS
1 Carcinoma
2 Sarcoma

## I ULCERS DUE TO ARTERIAL LESIONS

A. **Occlusive Origin.**—Ulcers due to obliterative arterial disease such as arteriosclerosis thromboangiitis obliterans or diabetes mellitus are the result of a failure of the arterial circulation to a part with consequent slough These occur most often at the periphery toes tips of fingers and over the heel They may be precipitated secondarily by some slight trauma Avascular necrosis occurs thereafter and these areas fail to heal Infection complicates the picture The organism usually is the streptococcus or the staphylococcus Fungus infection is a frequent contaminator The infection causes the pain and its control often determines the outcome

The lesions of *thromboangiitis obliterans* have been discussed on page 160 Typical ulcers appear either on the extremities or around the ankles These ulcers are extremely painful and in many instances the patient when seen already is addicted to some drug for relief of his pain There is an effort to heal along the edge with continuing sloughing in the center If the condition extends subcutaneous tissues and tendons become involved and if these are secondarily infected the part may be lost Occasionally ulcers due to thromboangiitis obliterans may heal spontaneously due to the remissions which are not unusual in this disease

The ulcers due to *arteriosclerosis* or *atherosclerosis* may be a primary type or secondary to some trauma.

Where the *arteriosclerotic lesion* is secondarily complicated by diabetes mellitus the prognosis is not as good because of the increased tendency to infection brought on by the diabetic state Local pressure alone also can cause arterial occlusion

**Treatment.**—Certain basic therapy applies to all ulcers (see Table 47) The treatment of ulcers following arterial occlusion has been discussed under the treatment of arteriosclerosis and thromboangiitis obliterans In addition to the treatment of the underlying cause, the basis of therapy is shown in Table 47

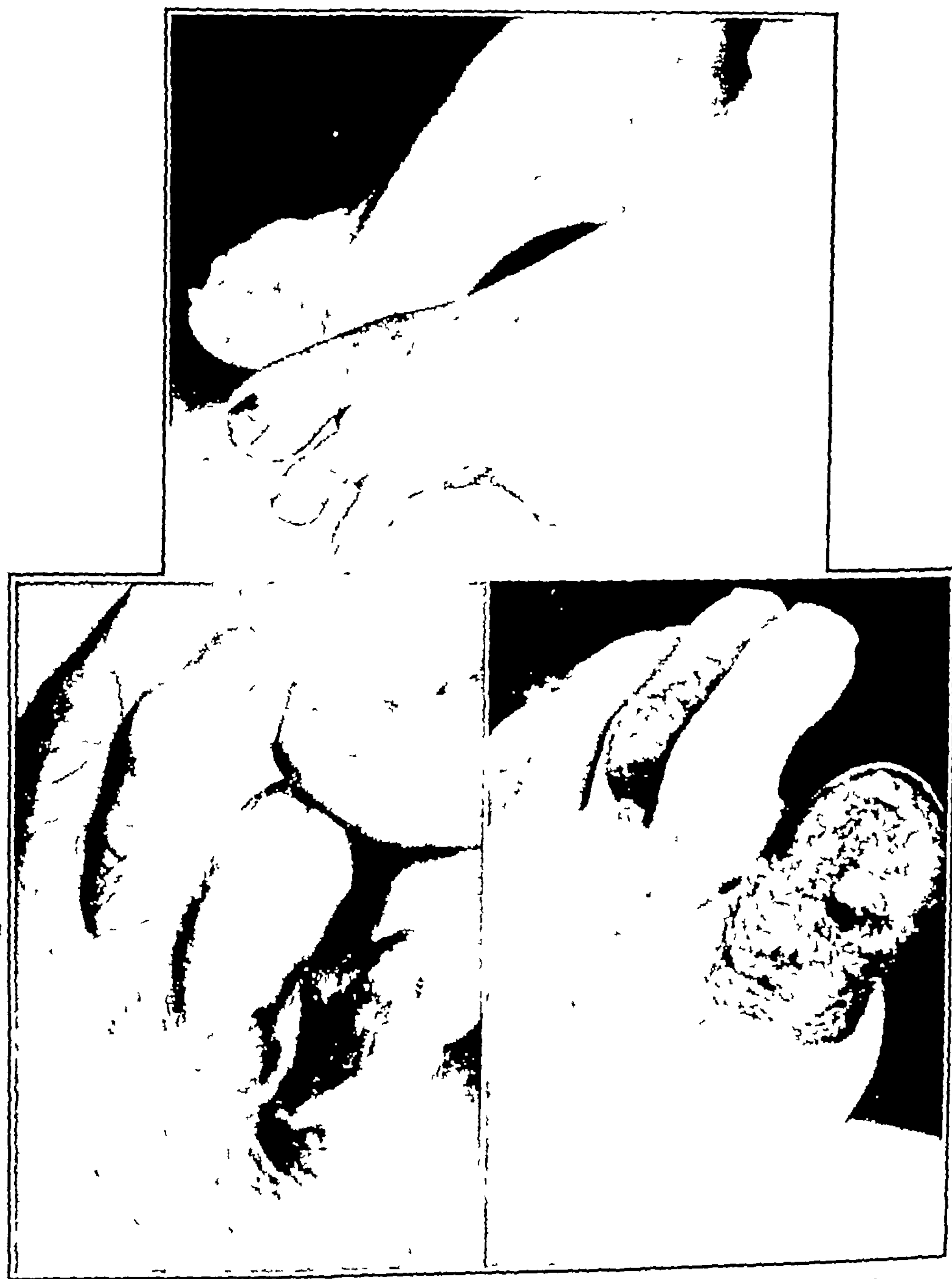


Fig 248 —Ulcers of arterial occlusion origin Arteriosclerotic ulcers due to occlusion of an artery

**General Treatment** —The general treatment of the underlying disease must be started See pages 162 to 176 Such systemic treatment must be active and continued It consists first of elimination of tobacco and all of the other spasm-producing drugs or factors The importance of

the avoidance of tobacco or any drug such as ergot or adrenalin, which cause increased spasm cannot be overemphasized

Second careful attention must be paid to the hygiene and the prevention of any irritation or further skin breaks

Third efforts should be made to stimulate the collateral circulation Medically this may be accomplished by warm baths administration of drugs mentioned in the chapter on Arterial Occlusion postural exercises,




FIG 249 —Ulcers of arterial occlusion origin. Thromboangiitis obliterans Slough of tissue supplied by a thrombosed artery

and temporary interruption of the sympathetic system The local circulation can be improved by interrupting the sympathetics in most instances This has been proven innumerable times While the value of this therapeutic measure in occlusive arterial disease has been questioned by some our experience leaves no doubt whatsoever of the value of the operation in over 90 per cent of the patients. Our feeling on this type of therapy is as strong as it is on the value of appendectomy in acute appendicitis Though all ulcers do not heal and sometimes a limb is lost thereafter, our Clinic

believes that this is due to delayed application of this treatment, continued smoking afterwards, or faulty technic This latter point may be due to technical, ineffectual or insufficient sympathectomy, or traumatic thrombosis or embolism at the time of the sympathectomy This basic

TABLE 46 —DIFFERENTIAL DIAGNOSIS OF ULCERS OF OCCLUSIVE ARTERIAL DISEASE

| | *Arteriosclerosis* | *Arteriosclerosis complicated by diabetes* | *T A O* | *Local Pressure* |
|---|---|---|---|---|
| Age | Over 40 | Over 25 | Between 20 to 40 | No relation to any age |
| Sex | Males 60% | Less of factor | Males 99% | No relation to any sex |
| Location | Toes—foot | Toes, foot, heel | Toes—foot | Heel, plantar surface, toes, malleoli, etc |
| Size | Varies | Varies | Varies | At site and size of pressure |
| Description | Necrotic, deep, involves tendons | Necrotic, often infected | Necrotic, inflamed | Necrotic, penetrating |
| Discharge | Slight until infected | Slight until infected | Purulent discharge | Slight |
| Edema | None | None | 30% (with thrombitis) | Depends on site |
| Pain | Moderate to severe | Greater with infection | Severe pains and rest pains | Mild |
| Claudication | Usual | Usual | Usual | Rare |
| Pulsation of arteries | Pulseless or diminished in 95% | Diminished in 95% | Pulseless or diminished in 95% | Palpable |
| Color changes | Pallor on elevation Rubor on dependency | Pallor on elevation Rubor on dependency | Pallor on elevation Rubor on dependency | None or slight |
| Spasm test | Less response | Respond in young | 65 % | Respond well |
| Veins | Normal | Normal | Superficial thrombophlebitis in 30% | Normal unless obstructed |
| Etiology | Arteriosclerosis, obliteration of arteries ischemia of tissue, trauma of physical, chemical or thermal origin | Same and diabetes | Arterial occlusion with thrombitis in 30% | Prolonged pressure, impaired nutrition of tissue Debilitating diseases |

therapeutic tenet is shared by most specialists in the field We believe that in all these patients with ulcers due to arterial occlusion sympathectomy is indicated A reduced lipid intake in the diet, avoidance of overuse of the part and the use of antithrombotic drugs have their place in therapy

These therapeutic principles are the same for all occlusive arterial diseases In addition skin grafts may be needed For such details see the chapter on Skin Grafting page 741 Other surgical efforts to increase the circulation by removing or by passing blocks have been discussed on pages 198 to 212 If the cause is an arterial aneurysm its primary care will be necessary to effect healing

**Local Treatment.**—The local treatment of ulcers following arterial occlusion consists mainly in keeping the area clean and preventing undermining and extension of the process. This treatment should be thorough, but great harm can be done by overzealous local therapy We no longer use chemical dyes and antiseptic solutions or paints Most ointments dyes, and drugs even though they may have a bactericidal effect are harmful in these lesions because they injure the tissue

TABLE 47 —BASIC PRINCIPLES OF SURGICAL TREATMENT OF ALL ULCERS

1 *General*
- A Eliminate or reduce the pathology of the cause (Arterial lesions improve the arterial circulation Varicose ulcers eliminate the varicose vein )
- B Overcome any secondary infection with antibiotic therapy based on sensitivity test
- C Improve patient s general status, i.e in the diabetic obtain diabetic control

2. *Local*
- A Hygiene keep the area clean remove fungus contamination and avoid injury to tissues,
- B Drain localized infections and use topical antibiotics
- C Prepare ulcer base for primary healing or skin grafting Remove slough without pain or bleeding Surgical or biological débridement. Avoid caustics or antiseptics which destroy tissue
- D Antithrombotic therapy in selected instances.

A routine treatment of ulcers following arterial occlusion is as follows

1 *Saline Soaks* —The affected part is immersed for one hour twice a day in a sterile saline soak. The temperature of this soak is maintained carefully between 88 and 92° F It is important not to burn the patient These soaks mildly stimulate vasodilation and remove excess drainage or slough from around the ulcer without trauma. Salt solution will not injure the tissue itself It has the benefits of a wet dressing without the disadvantage of the tissue cooling or maceration which occurs when a constant wet dressing is maintained

2 *Fungicides* – Inasmuch as all of these patients have a fungus contamination in the ulcer the affected part is immersed in a soak containing a mild fungicide potassium permanganate in a strength of 1 15 000 to 1 25 000 Immersion of the part in this solution for twenty minutes each day controls the fungus although it does not eliminate its growth Such soaks should be used in all cases unless there is a specific antipathy to the drug

Some of these infections spread They have a malodorous discharge and are caused by a mycotic infection secondarily contaminated by the pro-

gressive infection The use of potassium permanganate soaks helps many of these patients

The salts of the higher fatty acids such as propionic acid, undecylenic acid, and caprylic acid may aid in controlling the fungus[20] The sulfonated detergents with Hexachlorophene (pHisoHex and others) have a part to play These substances have a specific bactericidal and, possibly, fungicidal effect

3 *Local Débridement* —After the saline soak each day, any loose purulent or slough tissue should be carefully teased away from the ulcer with a smooth forceps *This should not cause pain or blood letting* If it is done painlessly, and without any blood appearing, normal tissue has not been injured Special attention should be paid to the edges so that there is no undermining If there is *marked* undermining, it may be necessary surgically to excise the overhanging tissue in order to avoid the pocketing

4 *Enzymatic Débridement of Ulcers* —Despite the fact that John Hunter mentioned that dead tissue could be digested away by enzymes as early as the 18th century, little or no attention was paid to it until 1949 when the therapy was revived for burns Trypsin[19] and other physiologic enzymes will destroy fibrinous strands and surface coagula in a matter of minutes The reaction of the tissue is an outpouring of serous exudates Organisms disappear and clean granulation tissue develops These substances work best in a pH of 6 8 to 7 5 They are applied in Sorenson's Phosphate Buffer solution which has a pH of 7 1 They must be used with care On ulcers, a powder base may be applied In the diabetic, ½ per cent solution of Tryptar[19] has been applied every four hours These substances have potential therapeutic value and their correct place in the treatment of ulcers will be developed with time

5 *Local Antibiotic Therapy* —The ulcer should be cultured and the correct drugs prescribed for the organism present, dependent on sensitivity Streptococci are most often found in such cases Antibiotic therapy is indicated both parenterally and locally It has been found that penicillin will eliminate the streptococcus from such ulcers if applied locally for a period of three days This antibiotic is indicated unless there is a reaction to it It is generally applied in a concentration of 300,000 units to 250 or 500 cc of saline solution

6 *Skin Grafts* —In rare instances, some of these ulcers may require a skin graft, but usually, where there has been an arterial occlusion, the recipient site will not be satisfactory for a skin graft In a few patients it has been possible to excise the base of such ulcers and achieve healing with sliding or split-thickness grafts using an electric dermatome[7]

7 *Surgery* —Other measures to increase the blood supply have been discussed on pages 198 to 212 Some ulcers progress despite therapy Others require local or major amputation See pages 234 to 255 for the handling of this problem

B **Spastic Ulcers** —In the repeated and continued spasm of blood vessels of a severe nature, the blood supply to the part may be affected to such an extent that ulcers will develop These spasms occur in those susceptible to some stimulus As the result of this stimulus the involved vessel goes into spasm The stimulus may be tactile, thermal, emotional or allergic

If the spasm does not relax ischemia and death of the tissue supplied by that vessel may occur with slough and ulceration This condition is seen more frequently on the upper extremities A typical example is the gangrene of the fingers seen in those patients who are susceptible to cold The ulcers of Raynaud s disease are another example

**Treatment.**—The surgical care of these spastic ulcers requires treatment of the underlying condition In many instances the treatment of the basic pathology is not too satisfactory or effective See pages 256 to 309 In

TABLE 48 —ULCERS OF SPASTIC ARTERIAL DISEASE

| | *Diagnosis* | | |
|---|---|---|---|
| | *Raynaud's Syndrome* | *Scleroderma* | *Pernio* |
| Age | Under 40 | Under 40 | Under 30 |
| Sex | Female | Female | Female |
| Location | Tip of digits | Tip of fingers toes, heel joints | Dorsum of foot or hand lower third of leg |
| Size | Small | Small | Varying size usually multiple |
| Description of ulcer | Sharp edges—round—superficial | Sharp edges—superficial | Indurated edges—reddened surrounding area |
| Discharge | Slight | Slight | Slight |
| Edema | None | None | Slight or none |
| Pain | Very painful | Very painful | Severe knife-like pain |
| Pulsation of arteries | Palpable | Palpable | Palpable |
| Veins | Normal findings | Normal findings | Normal findings |
| Spasm tests (Landis reflex dilation anesthesia nerve block) | Increased skin temperature | Increased skin temperature | Increased skin temperature |
| Capillary microscopy | Dilation of skin capillaries | Obliteration of skin capillaries | Normal capillaries |
| Etiology | Abnormal arteriospasm when exposed to cold or emotion etc | Unknown | Associated with cold weather Angiitis of smaller vessels |

some patients the elimination of all stimuli that cause spasm is sufficient to heal the ulcer In others it is necessary to change the individual s employment or climate especially in those allergic to low temperatures In Raynaud s disease sympathectomy when adequately performed will prepare the patient again for a useful life

*Local treatment* consists of keeping the part clean preventing undermining eliminating infection protecting the part from trauma and at times where the ulcer involves bone local amputation The conservative treatment is carried out in these cases as long as it is possible to do so or while there is some response unless there is a spreading infection

TABLE 49.—OCCLUSIVE AND SPASTIC ARTERIAL ULCERS

*Treatment*

| | *Medical procedure* | *Surgical procedure* |
|---|---|---|
| Rest | Bed rest | |
| Arterial dilatation | Controlled heat<br>Oscillating bed | Local nerve block<br>Paravertebral sympathetic nerve block |
| In selected cases | Drugs (spasm relaxers—Etamon-Priscoline-Dibenamine)<br>Mecholyl by iontophoresis<br>Hormones | Spinal anesthesia as test<br>Sympathectomy |
| Local | Soaks, saline and antibiotics for infected areas<br>Mild fungicides (potassium permanganate 1 15,000 to 1 20,000 solution)<br>Physiologic débridement | Débridement<br>Drainage when indicated<br>Excision of ulcer<br>Skin grafting |
| General | Treatment of diabetes, polycythemia, etc<br>Use of antibiotic drugs | Amputation if medical measures fail<br>Refrigeration |

| | Many ulcers heal spontaneously Therefore | |
|---|---|---|
| IMPORTANT | DO NOT use necrotizing substances—<br>1 acids<br>2 strong antiseptics<br>DO NOT use excessive<br>1 heat<br>2 cold<br>3 pressure of any type | Untimely or radical surgery may lose the limb or the life |

## II ULCERS DUE TO VENOUS LESIONS

Venous ulcers are of two main types varicose vein ulcers and thrombotic ulcers Trauma may be an incitor or aggravant in either type

1 **Varicose Vein Ulcers.**—These ulcers develop as a result of the pathologic processes following varicose veins with incompetent valves Blood is able to be returned from the lower extremities to the heart, an average distance of 4½ feet, through the venous system only by a system of vein valves These valves permit blood to rise against gravity In the superficial vein such valves are exposed to stress and strain and acute and chronic trauma They are not protected or supported by the muscles which surround the deeper veins Their failure is not unusual therefore, and when

e is a constitutional or inherited tendency to weakness the incidence of e failure is higher As the valves fail and the veins dilate there is a itant back pressure in the veins particularly in the greater and lesser ienous tree There is subsequent edema and a deposit of hemosiderin to the breakdown of blood cells in the area As the result of this back ·, the vascularity of the part is poor the skin becomes unhealthy and a or trauma or at times no trauma at all will cause a skin break The




Fig 250 —Ulcers due to varicose veins. Note secondary skin changes

n break is a most difficult one to heal These ulcers occur in 10 to 20 per t of patients who have varicose veins Leading from the ulcer are usu · one or more dilated tortuous feeding veins These ulcers frequently are nplicated by fungus infection and also secondarily infected Strepto- ci or staphylococci are the organisms that are cultured most often from h ulcers

The ulcers persist as long as the incompetent veins are present They ı be healed temporarily by conservative measures whenever the veins

are temporarily occluded by elevation or bandages. The causative pathology is relieved partially in this way, and the secondary ulcer tends to heal. This is unfortunate at times, as many patients believe that the incomplete and temporary treatment is a "cure." If one puts such a patient in bed with the leg elevated, this again collapses the veins and the ulcer usually will heal. A temporary healing or improvement of the ulcer will follow the use of any type of paste or boot support which collapses the veins at the same time. This leads to many "fad treatments" of ulcers. Some doctors attribute the healing that follows any compression of the causative veins to some "magic" ointment or type of bandage with which they produce the compression rather than the support itself.

**Ambulatory Preparation of Ulcer Site.**—In some patients it is necessary because of their economic status that the initial therapy to prepare the ulcer for operation be performed while the patient is on an ambulatory basis. The ulcer can be prepared by a combination of soaks and bandages with adequate support. The support which occludes the dilated veins achieves the healing. The bandages may be in the form of a boot. Such boots prevent excessive secretion by combining powders of which kaolin, bismuth subgallate and zinc stearate are examples. Some add a mild antibiotic, which helps if the patient is not sensitive to it. These boots are most successful where the skin disease is mild and infection is not a serious complication. Lassar's zinc oxide paste with 2 per cent crude coal tar makes a successful ointment.[8] The use of blood cells or blood gel paste may have some value locally.[2,9,10,22] Some of the fungicidal drugs or the salts of the higher fatty acids may be incorporated.[20] Before applying a pressure dressing one must be certain that the deep veins are functioning satisfactorily. This bandage works by support and the contraction of the skeletal muscles. It is safer to apply it as a supportive rather than a pressure bandage. This eliminates possible damage to the deep venous circulation or the arterial supply. A foam rubber sponge may be incorporated directly over the ulcer area to further restrict the venous and support the lymphatic drainage near the ulcer itself. The patient should be instructed not to walk if pain develops and to cut off the boot at once if it becomes tight. These ulcers recur when the support is removed and will continue to do so until the cause is eliminated.

The treatment of varicose vein ulcers has been discussed in this chapter on page 776. This treatment is twofold. It is aimed first at elimination of the complications of the ulcer including infection, and second, to eliminate the causative veins.

**Venograms for Diagnosis of Incompetent Vein Valves.**—These venous ulcers, whether primarily of varicose or thrombotic origin, usually are complicated by an inflammatory clotting when they are seen by the surgeon. If the original pathology was a deep vein closure, the surgeon must determine that there is an adequate deep vein return of blood before he eliminates the superficial system even though the latter is pathologic. In the past this decision has been made clinically by a *modified* Perthes test or by temporary occlusion of the superficial femoral vein at the operating table. In general, these tests work well. The present venography technic in experienced hands, however, will demonstrate the exact site of the in-

competencies It will determine whether these are in the saphenous system alone in the perforating branches or in the deep veins Where the experienced interpretation is available venograms are advised prior to extensive venous surgery See chapter on Roentgen Ray Visualization pages 730 to 731 and Postthrombotic Ulcer page 676

**Vein Resection for Varicose Ulcer** —Where there are incompetent valves the operation described on page 577 is the one of choice This operation should be performed if the ulcer is not inflamed This procedure includes




Fig 251 —Varicose ulcer *A* eight years' duration *B* four days after resection and stripping of saphenous vein *C* three weeks after operation (Pratt courtesy of Jour Am Med Assn.)

high resection of the saphenous vein and all of its branches at the femoral bulb resection of each incompetent point resection of the lesser saphenous vein if it is dilated or involved resection of any feeding veins just above the ulcer and suspension of the vein to the fascia. The vein is stripped between each blowout point

Occasionally an ulcer will heal after a local injection of the feeding vein but this is only a temporary expedient and resection will be necessary Subsequent to operation any dilated veins should be injected locally

**Local Treatment.**—The ulcer should be surgically clean before any operation is performed This can be accomplished, as outlined under the treatment of arterial ulcers, with saline and potassium permanganate soaks, antibiotic therapy locally and generally, elevation of the part, exposure of the ulcer in a vascular cradle at room temperature and muscle adenylic acid [18] All ointments and dressings are removed and the part is exposed in the cradle to the air If there is drainage, the part can be kept on a sterile towel, but the soaks usually will eliminate this rapidly Where the ulcers are necrotic they may be treated by some of the physiologic enzymes (See page 743 )

TABLE 50 —ULCERS OF VENOUS ORIGIN

*Diagnosis*

| | *Varicose* | *Phlebitic* | *Congenital* |
|---|---|---|---|
| Age | Over 30 | All ages | Normally young |
| Sex | Females predominate (66%) | No relation | No relation |
| Heredity | 80% | None | Predisposition |
| Location | Lower third of leg Medial aspect saphenous area | Lower third of leg Over internal-external malleolus | Lateral aspect |
| Size | Varies | Small | Varies |
| Description of ulcer | Irregular, shallow | Oval, round, deep, multiple | Irregular, shallow |
| Discharge | Scanty | Slight | Slight |
| Surrounding tissue | Brownish, bluish, scaly, indurated | Brownish, scaly, indurated | Brownish, scaly, indurated |
| Edema | Variable | Definite | Definite |
| Pain | None or slight | Very painful | None or slight |
| Pulsation of arteries | Palpable | Palpable | Palpable |
| Veins | Varicosities | Thrombosed femoral<br>Saphenous varicosities | Varicose aneurysm<br>Varicose tumors<br>Absence of valves |
| Etiology | Impaired venous drainage, valve failure, anoxemia, pigmentation, edema, dermatitis | Thrombophlebitis after injury, infection, systemic disease, operation, pregnancy, trauma | Congenital venous stasis, anoxemia, lowered resistance to trauma infection |

**Excision of Ulcer and Skin Grafting.**—Skin grafting is necessary in many of these ulcers but should be delayed until the causative veins are removed and the recipient site of the ulcer is surgically prepared for the skin grafting. When the skin grafting is performed, split-thickness grafts are satisfactory Brown's[7] electric dermatome has replaced all other instruments in our hands The dermatome of Padgett also is effective and supplies large, even grafts The old ulcer and its base (fascia) should be excised See the chapter on Skin Grafting, page 741.

2 **Postthrombotic Ulcers** —Such ulcers develop secondary to thrombitis (thrombophlebitis)

These ulcers were discussed in detail on pages 620 and 621 in the chapter on the Postthrombotic Syndrome They are extremely painful and usually occur approximately one year after a femoral phlebitis has developed Due to the thrombitis the valves in the superficial femoral vein have been destroyed A back pressure develops in this femoral vein



FIG. 252 —Malignant ulcer occurring on thrombotic leg Pathology melanoma This is a frequent site for such tumors

when it recanalizes similar to that present in an incompetent saphenous tree This back pressure is followed by edema skin changes dermatitis or skin breaks and ulcers. The saphenous system which was dilated to take over the function of the femoral vein when it was blocked becomes varicosed and has incompetent valves Its appearance is that of an advanced varicose vein pathology The correction of these thrombotic ulcers requires eradication of all the defective vein systems

The treatment of the phlebitic ulcer always is difficult because of its location and the advanced venous pathology which usually is present. Before any veins are ligated, a venogram is necessary to determine the adequacy of the deep veins and the sites of the incompetent valves. See page 778. The therapy consists of (*a*) surgical cleansing of the ulcer, (*b*) resection and stripping of the incompetent saphenous system, (*c*) ligation of the superficial femoral vein, if that vein is involved, has incompetent




FIG 253 —Massive spontaneous thrombosis *A* and *B*, Excision of thrombosis and entire saphenous vein, *C*, four weeks after operation

valves, and there is an adequate femoral profunda system.[16] In 15 per cent, excision of ulcers and skin grafts are required. In addition, 5 per cent require surgical sympathectomy to effect a cure.[15–17] The use of popliteal vein division, advocated by Bauer,[3–5] has not been more effective than superficial femoral vein resection in our hands except where venograms show this area to be the site of valve failure. Many of these patients require support for a considerable time. Some have been benefitted by an

inflated stocking [1] The importance of venograms has been stressed Ulcers due to congenital anomalies require excision of the causative vascular lesion or tumor

## III ULCERS DUE TO ARTERIOVENOUS LESIONS

Arteriovenous fistulas and aneurysms as well as arterial varices may cause ulcers For such lesions to be cured the abnormal artery vein connections must be interrupted Usually there have been many abnormal connections developed The area sometimes must be excised en masse See chapter on Arteriovenous Fistulas page 403 Skin grafts thereafter may be required

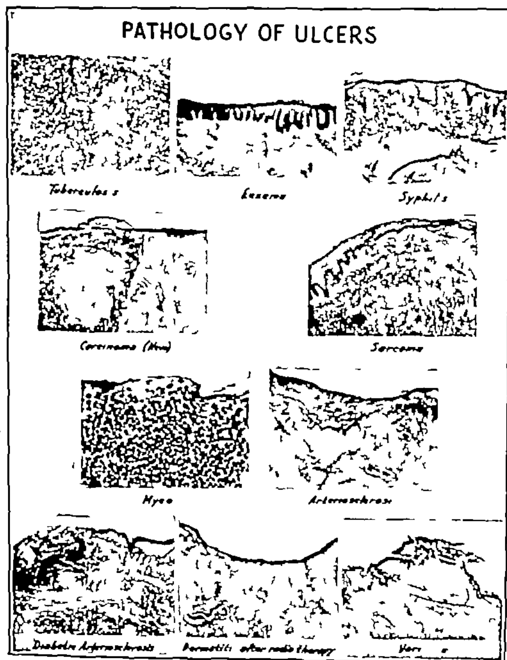



FIG 254 — Pathology of various types of ulcers.

## IV SPECIFIC ULCERS

**Syphilitic Ulcers** —Luetic ulcers are far from uncommon, despite the effective present day methods of treatment The population in large cities will show a 3 per cent incidence of syphilis Syphilis always should be considered in atypical ulcers A negative serological test is not sufficient to rule out syphilis In doubtful cases one should utilize the therapeutic test The treatment of such an ulcer is that of the underlying disease, although in most cases it is necessary to supply skin to the ulcer after the specific disease is controlled

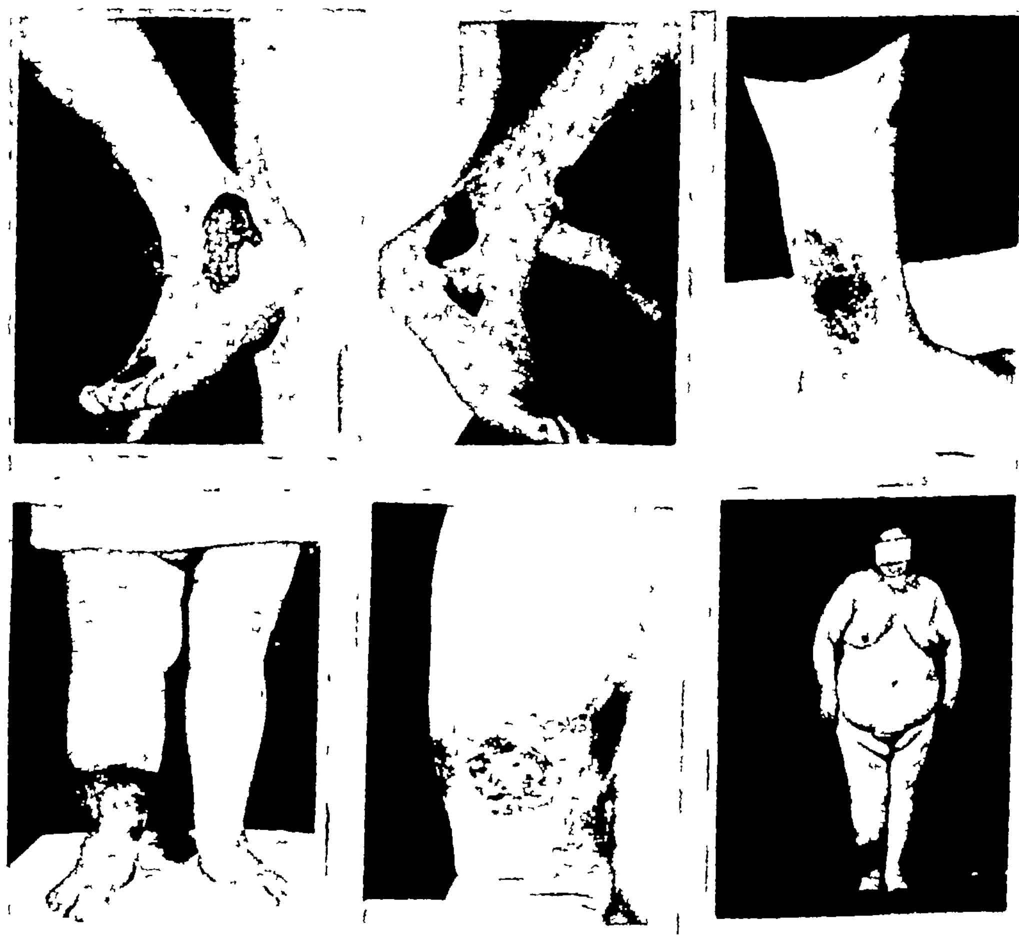


Fig 255 —Thrombitic (phlebitic) ulcers Note enlarged leg, obesity, hypoglandular status

**Ulcers From Drugs.**—Drugs such as the bromides, the arsenicals, and others, at times cause skin lesions which if neglected become ulcerated The treatment of ulcers due to drugs begins with the elimination of the incitant drug The restriction of the drug usually cures the ulcer The use of counterdrugs to overcome the allergy caused by the drug in question of course is obvious

**Ulcers Due to Vitamin Deficiencies.**—Ulcers follow vitamin deficiencies, especially of vitamins C, B and E. The treatment of such ulcers consists

of the administration of large doses of the vitamin which is lacking It may be necessary to give such vitamins parenterally as well as by mouth in high potency dosages. Ulcers due to vitamin deficiencies usually heal when the deficiency is corrected In some cases skin grafting may be required

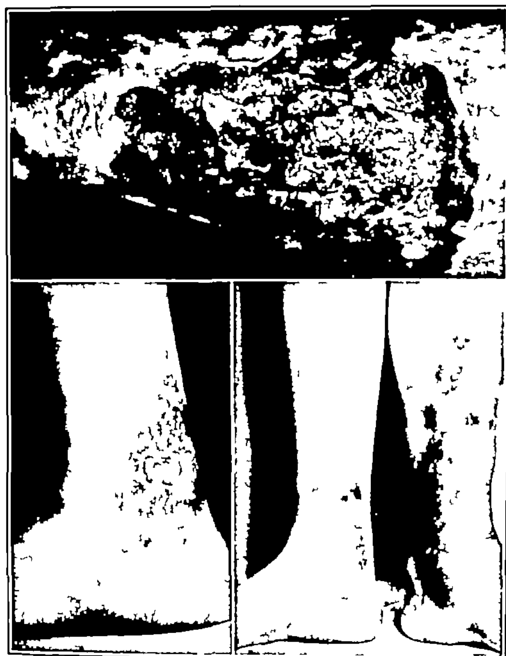


Fig 256.—Malignant ulcers These are not rare Any ulcer which does not respond to therapy should have a biopsy Upper ulcers due to carcinoma lower ulcers due to sarcoma

Each ulcer must be cultured and the sensitivity of that organism to the available antibiotics determined The antibiotic is then given in therapeutic dosages both generally and locally This needs emphasis since delay in selecting the right antibiotic may cause serious and extensive pathology




…67 Luetic ulcers. Approximately 3 per cent of the population* is infected … philis. Serology should be reported in each case. Resistant ulcers warrant a … ic test.

* …cities.

TABLE 51.—SPECIFIC ULCERS OF THE LEG

| | Diagnosis | | | |
|---|---|---|---|---|
| | *Lab. Positive* | *Usual Location* | *Description of ulcer* | *Etiology* |
| Syphilitic | Wassermann, Kahn or Kolmer | Medial aspect | Deep punched-out, raised indurated edges. Necrotic slough. Offensive odor | Lues, third stage |
| Tuberculous | X-ray, tuberculin test. Tuberculosis elsewhere, positive culture, biopsy | Middle-medial aspect of lower leg, preceded by painless nodules | Irregular undermined margins, grayish slough. Watery discharge. Crab apple nodules | Tuberculosis |
| Mycotic | Bacteriology | Toes and foot webs, unilateral | Raised border, small sinuses, purulent discharge | Fungus infection, etc. |
| Drug | Blood, stool and urine | Lower leg, multiple | Large, deep, round edges. Slight discharge if infected, pruritus, rash | Bromides, phenobarbital, iodides, arsenic, etc. |
| Vitamin deficiency | History, urine and blood | Lower third of leg | Superficial purpura, capillary fragility, hemorrhagic granulations | Avitaminosis C, B, E |
| Neurotrophic perforating | Specific tests | Sole of foot. Perforates to head of metatarsal | Perforation, suppuration involving bone and joints. Painless | Nerve lesions, tabes, syringomyelia, beri-beri, leprosy, diabetes, peripheral neuritis |
| Infections | Bacteriologic | Lower leg | Inflamed, undermined, serpiginous border, granular base, discharge | Various organisms. Often strept. |
| Gout | Blood chemistry and x-ray | Toes | Tophi ulcerate; ulcer base has chalky sodium urate deposit | Faulty metabolism of uric acid |
| X-ray, radium, isotope | Biopsy and Geiger counter, history | Any | Dermatitis, skin break, malignant | Burns |

**Other Specific Ulcers.**—Specific ulcers may develop from tuberculosis, actinomycosis, gout and other granulomatous conditions. Neurotrophic changes, deficiency of muscle adenylic acid and the exposure to x-ray, radium and radioactive isotopes may cause ulcers. Such ulcers usually are

not too difficult to diagnose if the possible cause is kept in mind A biopsy may be necessary

Treatment of such ulcers requires therapy of the underlying condition and often wide excision with skin grafting

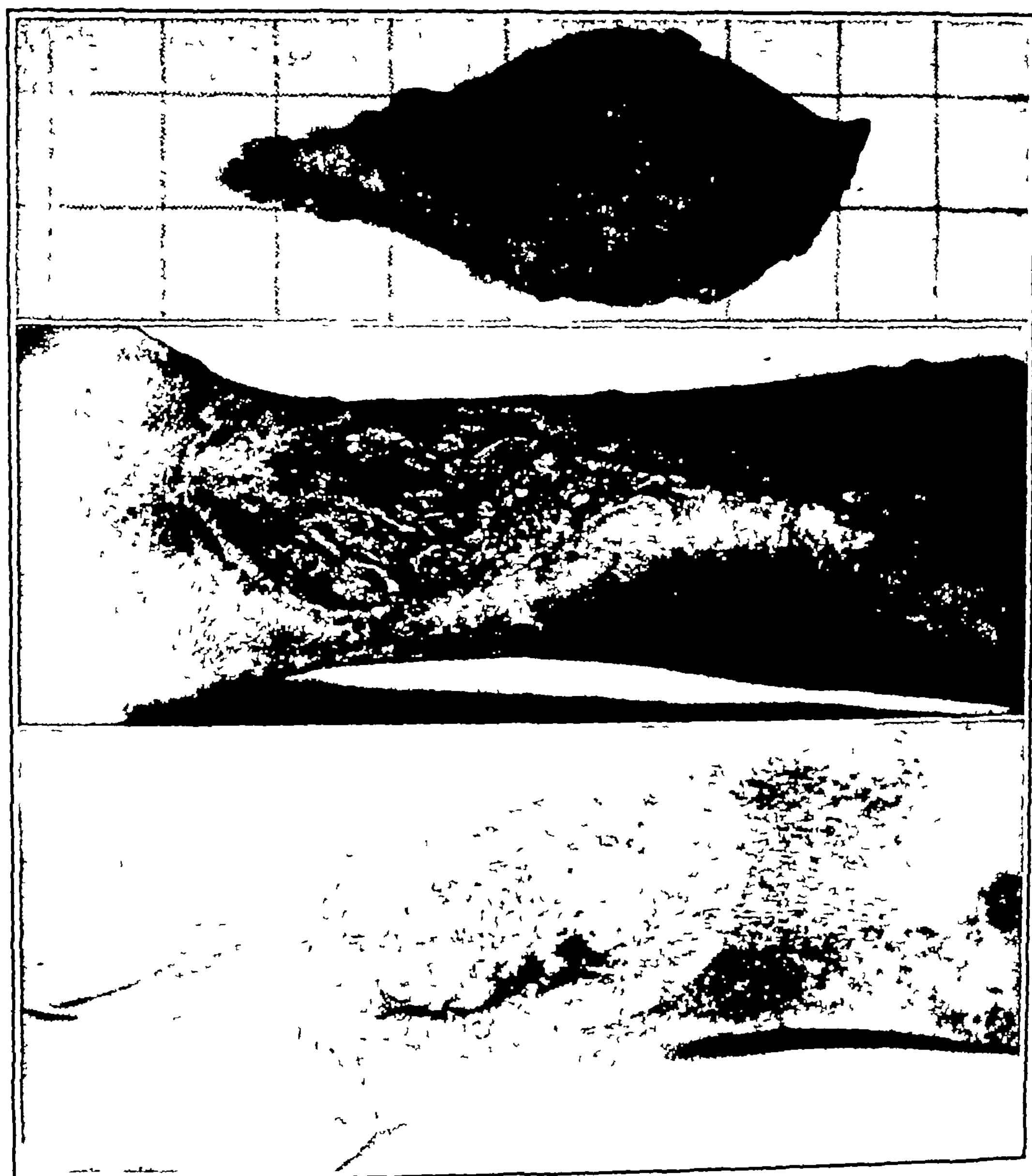


FIG 258 —Wide excision phlebitic ulcer with underlying fascia Sliding skin graft Healed skin graft

## V POSTTRAUMATIC ULCERS

The ulcers that follow trauma rarely are a specific vascular problem Their treatment consists of excision of scars, freeing of adhesions, mobilization of contractures, and at times reparative or substitute tendon or muscle surgery Skin grafts thereafter are a secondary procedure




Fig 259 —Ulcers due to *A* vitamin deficiency and *B* mycotic infection.

TABLE 52 —TREATMENT OF ULCERS OF VENOUS, POSTTRAUMATIC, SPECIFIC AND BLOOD DYSCRASIAS ORIGIN

| | *Medical Procedure* | *Surgical Procedure* |
|---|---|---|
| Rest | Elevation | |
| Venous drainage (avoiding stasis) | Support bandages, stockings, boots | Ligation and stripping |
| Local | Soaks: Saline<br>Antibiotics<br>Fungicides<br>Mecholyl by iontophoresis | *Débridement*<br>Incision and drainage (when indicated)<br>Excision of ulcer<br>Skin grafting |
| General | Specific therapy<br>Remove foci of infection<br>Antibiotics<br>Muscle adenylic acid | Surgical removal of cause<br>Sympathetic nerve blocks<br>Sympathectomy |
| Principles of healing | Cleanliness. Exposure at room temperature<br>Local vasodilatation<br>Avoidance of venous stasis<br>Elevation and support | Heal ulcer<br>Resection and stripping of pathologic veins<br>Resect ulcer and skin graft if necessary |




Fig. 260.—Ulcers may be due to many causes. Tuberculosis and the other granulomata are more rare. Anemia and drug ingestion may be a cause. *A*, Tuberculosis; *B*, Drugs Bromides; *C*, Anemia; *D*, Polycythemia.

## VI BLOOD DYSCRASIA ULCERS

The ulcers following specific blood diseases such as polycythemia anemia and leukemia must be treated medically as to cause Skin grafts are necessary in proportion to the amount of tissue lost



281 —Ulcers due to infection Treatment with antibiotics and skin graft.

## VII MALIGNANT ULCERS

Malignant ulcers are not uncommon In the Vascular Clinic of St Vincent's Hospital, ulcers have been referred to us diagnosed as of arterial or venous origin which were later proven malignant We now biopsy all ulcers that do not respond at once to therapy.

**Treatment** —The treatment of a malignant ulcer depends on the type and the extent of the lesion Surgical excision is the preferred treatment In some cases, amputation may be necessary Roentgen rays and radium, with secondary grafting, may be effective in some

Where the growth is not highly malignant, excision of the ulcer and grafting with the electric dermatome have arrested the process

## VIII ULCERS DUE TO INFECTION

Ulcers may arise due to local infections Certain strains of the streptococci have a specific action on the skin, seeming to digest it away Other organisms, perhaps in symbiosis with the streptococcus, have similar effects, the colon bacillus often being an offender Certain fungus organisms alone or with others have this irritating and digesting action

**Treatment.**—Therapy depends on the organism involved In the aerogenous organisms the use of hydrogen peroxide is effective In the fungus group, potassium permanganate solution may help In others the chlorophyll preparations aid In some, the gallic acid preparations are of value [20]

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Chapter

# 44

# RADIOACTIVE ISOTOPES AND ATOMIC ENERGY IN THE DIAGNOSIS AND TREATMENT OF CARDIOVASCULAR DISEASES

## *The Effect of These Elements and Their Explosion on the Cardiovascular System*

THE radioactive age poses two medical problems. The first is the prevention and treatment of the destruction caused by this force when used as a bomb While this is of interest to all doctors in their role as guardians of the nation's health, and their functions in military medicine, it is not of primary interest to us as vascular surgeons The vascular system will be injured like other parts of the body, both by explosion and by burn The secondary effects of the rays will also present vascular problems These will be of more general surgical significance than they will be to the vascular surgeon

The second important phase of this radioactive problem is the utilization of radioactive substances in the diagnosis and treatment of certain diseases

In 1945 the atomic bomb explosions at Nagasaki and Hiroshima opened new vistas in destruction as well as in medical and surgical diagnosis and treatment At the same time these atom-shattering events posed a new problem in the treatment of injuries and burns heretofore unknown The effect of the bombings on the Japanese people was deplored by the citizens of the bombing nation only slightly less than it was by the bombed people Only the realization of the tremendous cost in life and living which the storming of the shores of the home islands of the Japanese Empire would have cost to both the attackers and the besieged can justify the savage results of the explosions That this terrific loss was averted, undoubtedly due to the observations of the effects of such bombings argues further for the justification of the employment of this agent The loss of the people of two cities and a war by the Japanese contributed in no small way to the progress of the world

Thus, the medical profession has in hand a potential source of power unknown before in this world This profession has learned to harness the power of the scalpel and roentgen ray and radium to act in the interest of the patient A like result with this new power is a goal While the medical possibilities of atomic energy diagnostically and therapeutically are boundless, they are still in the potential state

In the contrary vein, we have created the greatest destructive agent thus far known To first, second and third degree burns we now must add that

of far reaching and acting rays and secondary effect on various glands and organs We have opened a whole new field of prophylactic and active therapy necessary when the destructive action of this force may be released upon the world Even worse destruction is possible with the hydrogen bomb

It is difficult for the lay mind to divorce the destructive potentialities of the atomic bomb from the possible benefits medically and biologically which may accrue from radioactive isotopes It just happens that the same forces which make heavy elements fissionable make lighter ones radioactive It is only the combination of this production mechanism that in any way associates the two Artificially produced isotopes were used biologically many years before 1939 when the first atomic fission was reported

The direct clinical applications of atomic isotopes in clinical medicine so far are few Three outstanding ones are examples

1 Radioiodine has a place in the diagnosis and treatment of thyroid disease and in overfunction or growth of the thyroid

2 Radiosodium through its tracer action has a place in the study of vascular diseases

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1 Radiocarbon as an indicator in the degradation and the re-synthesis of carbohydrate and fat

2 Heavy nitrogen in the study of the dynamic equilibrium in the protein pool. This is of importance in the metabolism of tumors

3 Radioiron to determine the bone marrow function in septic states

4 Red blood cells tagged by radioiron to measure the red cell mass in burns

5 Radiosodium to determine the extracellular phase in shock infection and burns as well as the peripheral circulation

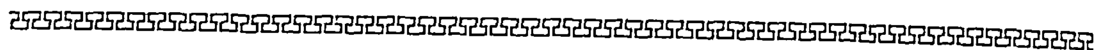
6 Radiopotassium to measure body potassium and cell permeability

7 Heavy water transfer to indicate the total body water and water exchange

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From a review of the literature the report of the Council on Pharmacy and Chemistry the data submitted by the Council on Physical Medicine the United States Atomic Energy Commission and the reports from the Department of Defense considerable data have been assembled At this stage the following can be stated regarding the use of radioactive substances in vascular diseases

**The Source of Neutrons** —A radioactive isotope has an atomic nucleus which differs from a non radioactive one because of an extra neutron This neutron does not affect the chemical action of the atom in most reactions The nuclear changes result in the instability of the atom which may disintegrate suddenly This disability varies from one isotope to another



Chapter

# 44

# RADIOACTIVE ISOTOPES AND ATOMIC ENERGY IN THE DIAGNOSIS AND TREATMENT OF CARDIOVASCULAR DISEASES

## *The Effect of These Elements and Their Explosion on the Cardiovascular System*

THE radioactive age poses two medical problems The first is the prevention and treatment of the destruction caused by this force when used as a bomb While this is of interest to all doctors in their role as guardians of the nation's health, and their functions in military medicine, it is not of primary interest to us as vascular surgeons The vascular system will be injured like other parts of the body, both by explosion and by burn The secondary effects of the rays will also present vascular problems These will be of more general surgical significance than they will be to the vascular surgeon

The second important phase of this radioactive problem is the utilization of radioactive substances in the diagnosis and treatment of certain diseases

In 1945 the atomic bomb explosions at Nagasaki and Hiroshima opened new vistas in destruction as well as in medical and surgical diagnosis and treatment At the same time these atom-shattering events posed a new problem in the treatment of injuries and burns heretofore unknown The effect of the bombings on the Japanese people was deplored by the citizens of the bombing nation only slightly less than it was by the bombed people Only the realization of the tremendous cost in life and living which the storming of the shores of the home islands of the Japanese Empire would have cost to both the attackers and the besieged can justify the savage results of the explosions That this terrific loss was averted, undoubtedly due to the observations of the effects of such bombings, argues further for the justification of the employment of this agent The loss of the people of two cities and a war by the Japanese contributed in no small way to the progress of the world

Thus, the medical profession has in hand a potential source of power unknown before in this world This profession has learned to harness the power of the scalpel and roentgen ray and radium to act in the interest of the patient A like result with this new power is a goal While the medical possibilities of atomic energy diagnostically and therapeutically are boundless, they are still in the potential state

In the contrary vein, we have created the greatest destructive agent thus far known. To first, second and third degree burns we now must add that

of far reaching and acting rays and secondary effect on various glands and organs We have opened a whole new field of prophylactic and active therapy necessary when the destructive action of this force may be released upon the world Even worse destruction is possible with the hydrogen bomb

It is difficult for the lay mind to divorce the destructive potentialities of the atomic bomb from the possible benefits medically and biologically which may accrue from radioactive isotopes It just happens that the same forces which make heavy elements fissionable make lighter ones radioactive. It is only the combination of this production mechanism that in any way associates the two Artificially produced isotopes were used biologically many years before 1939 when the first atomic fission was reported

The direct clinical applications of atomic isotopes in clinical medicine so far are few Three outstanding ones are examples

1 Radioiodine has a place in the diagnosis and treatment of thyroid disease and in overfunction or growth of the thyroid

2 Radiosodium through its tracer action has a place in the study of vascular diseases

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and the disintegration time may be from a fraction of a second to several million years This stability cannot be changed by external factors, therefore the disintegration time of half of the atoms in any material containing isotopes is referred to as a "half-life" Those with the shortest "half-life" are safe for clinical use [9] Thus, radioactive sodium with a "half-life" of 14 8 hours can be given with the knowledge that in 3.7 days, only 0 625 microns remains in the body When the radioactive body disintegrates, emanations of beta, gamma or sometimes alpha and neutron rays are emitted The effects, biologically, result from the rays and not from the destruction of the molecules

*Types of Particles* —(*a*) *Alpha* —These are heavy and double-charged These two neutrons and two protons, identical to helium, produce local but intense damage, because they collide with other nearby atoms

(*b*) *Beta* —These particles are of high speed and have a penetrating power up to 32 for phosphorus They affect the immediate area of the molecule destruction

(*c*) *Gamma* —These rays penetrate more deeply and are similar to $x$-rays

(*d*) *Neutrons* —These rays penetrate deeply because they lack electric charge They produce local effect by collision with other rays The penetration of tissue depends upon the speed of the emanations Thus, low speed and energy neutrons are more subject to collision than high speed ones High energy beta particles penetrate deeply before ionization is produced. The depth is determined by the original energy of the electron Thus betatron can produce intense ionization at the depth desired and therefore is useful in the treatment of cancer

Neutrons can be obtained from (1) a cyclotron used to bombard a beryllium target with highly accelerated deuterons, (2) a mixture of radium and beryllium in which the alpha particles bombard the beryllium, and (3) the fission of uranium in the uranium pile and the atomic bomb

**Hazards in the Use of Isotopes.**—The danger to the patient and the investigator in using radioactive substances cannot be ascertained exactly Radiations affect certain body parts For example, leukemia occurs 10 times more often in radiologists than among other physicians [9] Thus, a check must be kept upon the blood-forming organs The safe amount of exposure is conjectural The effect of ingestion orally or by inhalation of radioactive isotopes may be serious In a seventy-year life, an individual receives from the cosmic rays 20 to 40 r An internal deposit adds 2 to 5 r A gastrointestinal $x$-ray series may add 75 r X-ray therapy for such a lesion as a skin carcinoma often is 5,000 r A fatal total body irradiation for a human is estimated at 400 r These figures are stated only to indicate the potential danger of exposure to the patient and to the user In the light of our present knowledge and with the equivocal results, it is apparent that use of these substances, diagnostically and therapeutically, should be limited to those in whom the beneficial effects of exposure far overcome the potential dangers In addition, isotopes having a "half-life" of more than thirty days should be used with full recognition of their hazard [9] The burn effects are minimal but the secondary ray damage may be great with the use of the substances therapeutically The Atomic Energy Commission

minimizes this danger by insisting that approved medical organizations must possess adequate facilities for assaying handling and disposing of the radioactive material as well as the clinical care of the patient

**Measurement of Radioactive Isotopes** —When radioactive substances split, they collide with other atoms producing ionization and the result is that free electrons are formed These can be measured by the Geiger counter the scintillation counter and the ionization chamber The Geiger counter thus records the disintegration of the atom The scintillation counter absorbs the radiation in a crystal which causes a minute flash detectable by a photomultiplier tube and amplifier It is the best recorder of the gamma ray emissions The ionization chamber is a simple device amplifying the rate of fall of an impressed charge between two electrodes

**Diagnostic Procedures.**—The tracing of a radioactive substance throughout the body, while not yet accurate may be of value in determining the rate of circulation As the radioactive substance reaches the periphery and enters the extravascular fluid from the capillaries a counter (see above) will show the rate of build-up of the radioactive substances in the extracellular fluids until they come into equilibrium with the plasma radioactive isotopes In this way to a degree the type and adequacy of the circulation present can be determined Less accurately the actual rate of circulation from the arm to the foot can be determined by this method

The counter registers the amount of radioactive substance *i e* sodium in the extravascular tissues and not in the arteries The tube must be carefully shielded to prevent random radiation from various parts of the body from being recorded Five cubic centimeters of solution containing 100 to 200 microcuries of radioactive sodium can be injected in the antecubital vein As the rate of the count suddenly rises the time of this increase is taken as the circulation time The variations in the circulation time to the extremities in normal individuals is so great that the results are not accurate So many uncontrollable factors influence the time of arrival of the sodium at the periphery that interpretation often is difficult

Elkin[1,3] and his co-workers tried injection of the radioactive substance into the gastrocnemius muscle and measured its rate of disappearance from the tissues as a means of determining the relative effective blood flow No changes were found in hypertension The removal rate of sodium in the occlusive arterial diseases was inconsistent and varied with emotional and postural changes In general, those with advanced disease showed a slower removal rate The patients with thrombophlebitis generally showed a slower sodium removal although again the results in any individual are difficult to interpret. It is not unlikely that other technics for determining circulation time using this medium will be found

*Radioactive sodium chloride* $Na^{24}Cl$ already has some place in the field of vascular surgery It has a short half life (14 8 hours) is nontoxic and is rapidly excreted Its use is restricted to areas close to the source It can be used with a body radion of less than one roentgen Radioactive sodium emits hard beta and gamma rays These are detected readily by electronic equipment outside of the body Radioactive sodium does not have long-continued radioactivity and is not harmful in small quantities It is an ideal substance therefore for investigative use

By the injection of radioactive sodium chloride and the extent of its permeation, the degree of ischemia of a part can be determined to some degree. This may be of use in some instances to determine the level of amputations.[2,3,9] Adequate circulation in a limb can be determined in a like manner. This test may be confirmative, for example, after an embolism when this fact cannot be determined clinically for a considerable time. Sodium 24 predominantly is an extracellular ion. Cardiac dynamics can be determined after its intravenous injection. The time required to obtain a constant level of radioactivity in a limb after injection of intravenous sodium 24 will aid in the determination of the circulation in that part. It may be of value to estimate the efficiency of medical management.

*Radioactive phosphorus*, $P^{32}$, has a "half-life" of 14.3 days and emits beta rays and may aid in the study of the peripheral circulation. Its penetration can be improved by the addition of a non-radioactive sodium phosphate. The possibility of its value in phospholipid metabolism exists, and it may be of value in the study of the chemistry of faulty lipid metabolisms, such as is present in arteriosclerosis. Its value in polycythemia appears definite. It is of value in measuring the circulation of an extremity, also, to determine red blood cell volume and possibly in the treatment of hemangiomas. (See above.)

Radioactive phosphorus has helped to locate brain tumors. It has been used to treat polycythemia and leukemia. Its biologic aspects have been mentioned above.

*Radioactive iodine*, $I^{131}$, which has already proven its value by its localization in the thyroid gland in thyroid carcinoma metastases, may have an effect either by its glandular action or directly on certain hypervascularized areas in a similar way to its effect on the thyroid gland. Iodine 131 emits beta and gamma rays and has a "half-life" of eight days. The beta emission has the inhibitory effect on the tissue. The gamma ray permits the determination of the amount and site of the isotope deposit. Radioactive iodine is available as carrier-free sodium iodide (5 mc. per ml.). It is given by mouth. Its retention in the body depends on the amount of iodine present and the thyroid gland activity. Up to 25 per cent will be fixed by the thyroid gland in the normal individuals. Fifty to 80 per cent will be held in the hyperthyroid, while in the myxedematous patient, the amount will be under 10 per cent. A follow-up study of nearly 400 patients treated with radioactive iodine for a hyperactive thyroid showed that 76 per cent of the patients obtained remissions with one to two doses. Approximately 15 per cent developed hypothyroidism. If exophthalmos was present, 20 per cent had regression, while 60 per cent had some degree of improvement. The 8 deaths reported do not appear related to the therapy. The treatment seems effective in selected instances.[9]

*Radioactive Carbon*, $C^{14}$.—Carbon 14 emits weak beta rays. It has a "half-life" of 5,600 years. Thus, it can be used on long-term studies and can be stored. This long life adds to its dangers, as once it is in the human body, its action is fixed irreversibly. Its experimental value at present has been its use in the determination of the effect of digitalis.[9]

*Other Radioactive Isotopes*.—Radioactive gold ($Au^{198}$ or $Au^{199}$) has been used for local irradiation. It emits both beta and gamma rays. It has been

used in leukemia. Radioactive arsenic so far has failed as a vascular therapeutic agent.[9] Radioactive cobalt may replace radium in certain conditions.

A summary of the possibilities of radioactive isotopes in diagnosis, therapy and research, as applied to the vascular system, is as follows. The outline repeats some points mentioned above.[1,2,3,4,5,6,9,11,12,13]

A. *Diagnosis*

1. Radiosodium chloride, $Na^{24}Cl$, can be injected intravascularly to study congestive heart failure, to study limitations of blood flow in ischemic areas, and to measure the water content of the body.

2. Sodium radioiodide, $NaI^{131}$, can be administered to study its localization in thyroid disease, especially in malignancy with metastases. It may also serve to distinguish, for instance, cretinism from mongoloid idiocy.

3. Compounds of radiostrontium, $Sr^{89}$, may prove useful in the diagnosis of tumors and diseases of bone and teeth, since they are deposited in the hard tissues of the body.

4. Compounds of radiocarbon, $C^{14}$, also appear in the bone as carbonate. This substance has a biologic use also.

5. Other possibilities include radioiron, $Fe^{59}$, radiocobalt, $Co^{55-60}$, and radiophosphorus, $P^{32}$.

6. Micropathologic methods may include direct photographic prints from thin sections of tissue that have accumulated radioactive elements.

B. *Therapy*

Radioactive isotopes are being tried in the treatment of myelogenous and lymphatic leukemia, lymphosarcoma, some lymphomata and polycythemia. They are of some value in Hodgkin's disease. Their greatest value at present is in the diagnosis and treatment of carcinoma and its metastases, particularly from the thyroid, breast and testes.[4,9,11]

1. Radioactive phosphorus, $P^{32}$, diffuses wherever ordinary phosphorus goes in the tissues, particularly in the phospholipids. Hence it appears in the red blood corpuscles and erythropoietic tissues. It has been utilized in various blood dyscrasias and has been found beneficial in polycythemia vera. Its help in tumors of the brain has been mentioned.

2. Radioactive iodine, $NaI^{131}$, has been tried in hyperthyroidism and in thyroid carcinoma with metastases. In the former condition the results have been reported good; in the latter condition, available information indicates that it not only determines the site of the metastases but has a beneficial treatment effect. Removal of the parent growth increases the concentration of the substance in the daughter growths.

3. $Au^{196}$, $Au^{198}$ and $Au^{199}$ (radiogold) have been tried in leukemia, lymphoma and Hodgkin's disease with dubious results.

4. Many possibilities remain to be explored. The list of isotopes available for medical use is increasing. Short lived isotopes disappear inconveniently fast; long lived isotopes like ordinary radium ("half-lives," 1,580 years) must be placed in the human body with due regard to the length of action and the possible ill effects. Radioactive carbon one day may replace radium. The rate of disappearance of a given isotope from the body depends on (1) its rate of radioactive decay, or "half life," and (2) its rate of excretion. These are quite independent of each other.

C *Medical and Biological Research*

1 Atom exchange If CO is mixed with $CO_2$ and then heated in the presence of a catalyst, one soon has a mixture that includes CO and $CO_2$ Thus atom exchange can be demonstrated for research purposes

2 Sometimes atom exchange fails to take place in the body It cannot occur if two molecules do not come sufficiently close For instance, the iron atoms in hemoglobin do not exchange with the iron atoms of the blood

3 Tagging experiments can be used in studies on the following

(*a*) The localization of normally occurring atomic species within the body, particularly within different parts of the cell

(*b*) The absorption of small amounts of medicaments, for instance, by the percutaneous route

(*c*) The localization of foreign atomic species in the body, especially poisons

(*d*) The course of hydrogen atoms in oxidations and reductions

(*e*) The course of carbon atoms during decompositions and synthesis

(*f*) The stability of particular compounds and linkages

(*g*) The behavior of carcinogenic and antibiotic substances

(*h*) The activity of viruses and bacteria

(*i*) Disorders of the heart and the effect of drugs on this organ

4 A special technic in which tracers are useful is that of "dilution" If a radioactive isotope R* exchanges freely with a stable isotope R# within the body, then one can determine how much R# the body contains by administering a known amount of R*, allowing time for equilibrium to be established, withdrawing a sample of the equilibrium mixture, and determining the ratio of R* to R# From this ratio, one can tell how much R# was initially present by calculations analogous to those used in determining blood volume by injections of known amounts of dyes like vital red

5 Effects of radiations

(*a*) Pathology of burns and of radiation sickness The measure of the red cell mass in burns can be determined by tagging red blood cells with radioiron as reported above This helps in determining hemoconcentration [11]

(*b*) Studies on genetics, eugenics, and dysgenics

(*c*) Production of experimental injuries as prerequisite for development or rational therapies

**III Effects of Atomic Explosions and Burns.**[4 7 8 16–18]—An atomic explosion generates enormous amounts of kinetic energy. Four-fifths of this is in the form of heat, infra-red and ultraviolet radiation While man's fear of the unknown has generated much thought on the radiation ray dangers, a more proper perspective requires acceptance of the fact that the primary medical problem in such explosions will be burns In a bomb the size of one used in Japan, the hypocenter of the bomb would be 1500 yards ($2\frac{1}{2}$ square miles), and destruction would be complete in this area In the area from 1500 to 4,000 yards (14 square miles), the survivors would be seriously burned Spontaneous ignition of clothes occurred as far as 3500 yards ($2\frac{1}{2}$ miles) from the point of explosion [4 16] These facts and figures must be multiplied on the basis of greater potential damage of the

modern bomb and the possibility of more than one bomb being detonated at the same time The medical problem that a hydrogen bombing would impose has not been met. The potential problem is awesome In similar manner it may be decreased by greater distance from the site of explosion This agent while primarily destructive is inconsistent in its effect Recent test explosions showed that some structures near zero center escaped destruction while those at a distance were pulverized Wind and other weather variables modify the effect as does the height of the explosion and the terrain We must understand the tremendous force with which we are dealing The bombs exploded 2 000 feet above these Japanese cities set off a chain reaction causing a fission of the uranium and plutonium atoms within a millionth of a second The energy released was equivalent to at least 20 000 tons of T N T (trinitrotoluene) The temperature rise[9] at the center of the explosion was greater than 1 000 000° C which is comparable to the temperature of the sun All the metal in the bomb vaporizes producing gases which at their high pressure, move out at such a speed as to cause a shock wave Thus the blast effect develops The high temperature produces the so-called 'ball of fire' at the center temperature of which is 300 000 ° C The rate of its dispersion is 100 miles an hour and the atomic cloud forms as the heat is dispersed and it may rise to 40 000 feet. Thereafter energy is released in the form of ionizing radiation the most serious of which is the gamma ray The day the bomb exploded over Hiroshima 300 000 people were in the city Eighty thousand died immediately or before they reached medical aid Forty thousand people were seriously injured [6,12]

Three types of injuries resulted The blast injuries from the shock wave affected 75 per cent of the casualties Sixty five per cent had burn injuries Only 15 per cent had radiation burns Ninety per cent of the blast casualties had contusions and lacerations The others so affected never came under medical care

Civilian defense is concerned with the control and care of the casualties which will result from atomic or hydrogen bombing Most of our facts figures and planning are based upon what occurred in the Japanese cities [9,13,14] The future bombs effect admittedly will multiply the primary and secondary destruction an unknown number of times It is hoped that preparation for such an explosion which was nil in Japan will decrease relatively the expected mortality and morbidity from such a catastrophe In any contemplation of the effects of a bomb one may disregard the central zone of total annihilation which will vary directly with the force of the bomb No amount of preparation can save those in that area. If there were 600 000 casualties it could be estimated that 200 000 would be killed outright or would not survive twenty four hours Of the 400 000 who survived the first day 100 000 would die within the next five weeks Of these 400 000 240 000 would have burns due either to the flash or the secondary fires Two hundred and fifty thousand including many of those with burns would have mechanical injuries from flying débris glass bricks etc. Over one-half of these would be shocked and this combined with the burn cases would mean that fluids to restore the circulating blood volume would be required in enormous quantities From our recent experiences it is certain

that whole blood could not be made available Plasma, gelatin and other replacements and salt solution would have to be the substitutes The problem becomes nearly insurmountable The plasma expanders are being stock-piled Fortunately, these substances can be reproduced rapidly One hundred and twenty thousand patients would suffer radiation injuries Their symptoms appear later and require specialized care Those interested in the care of injuries after an atomic explosion on a medical or surgical basis are referred to the many excellent treatises now available on the subject We are not interested in atomic energy blasts in this book, but only in the vascular lesions Any serious burn affects the vascular system The flash burn has a radius of 2 miles, and only those parts of the body directly exposed are affected These result in second and third degree type burns The flame burn is no different than the thermal burn in any fire The keloid scars following the burns in Japan probably were due to the poor nutritional state of the injured and the improper and inadequate care supplied The ionization radiation burn, occurring in only 15 per cent, is caused mostly by the gamma rays [8]

These points are mentioned only that the reader must have in mind the possible severity of explosion and the dangers attendant to the use of these substances diagnostically and therapeutically The secondary effects on the vascular system, as well as other systems, such as the generative one, is not covered in this discussion

In summary therefore, it can be said that the diagnostic and therapeutic potentialities of radioactive isotopes and atomic energy still are unknown The field has not been explored fully Better control of the substance must be attained and more experience is necessary in order to use it safely on patients Adequate protection of those in charge of the substance is fundamental The use of radioactive isotopes must be confined to well set-up laboratories in which a physicist trained in radioactive principles, excellent technicians, and careful supervision are present The potentialities of the destructive action on the vascular system of atomic explosions must be kept in mind At the present time we know radioiodine is of value in the diagnosis and treatment of thyroid hyperactivity Radiophosphorus helps in polycythemia and leukemia as well as the localization of brain tumors Radiosodium helps the study of vascular diseases The other radio substances such as carbon, potassium and many others are under study

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# SECTION IX

## Injury, Occupation in Cardiovascular Disease

## Chapter 45

## CARDIOVASCULAR DISEASE AND TRAUMA

*The Relationship of Trauma and Occupational Hazards to Cardiovascular Diseases*

In this discussion, the trauma incidental to the patient's work and the hazards of the occupation itself will be considered in their effect on the cardiovascular system This problem is discussed from the compensation and insurance standpoints The effect of direct trauma on blood vessels, such as occurs during war time or in severe industrial accidents, is covered in the chapters on Injuries to the Arteries, Injuries to the Heart and in the chapter on Varicose Veins under Ulcer and in the chapter on Pathologic Venous Clotting These points are not discussed again

Increased longevity and improved working conditions have been followed by the continuation of active work of a great many people beyond previously accepted retirement ages No longer is an individual considered elderly at sixty, and it is now recognized that gainful production is possible by many men and women up to seventy or seventy-five years of age The extension of the retirement age and its individualization means that in the future the employment rolls will contain an increasingly higher percentage of those previously considered aged This applies to both physical and mental employment, although the problems discussed in this chapter will apply most often to the former

With this change an older group of people, therefore, will be engaged physically in labor when they have cardiac or vascular lesions

If such individuals suffer an occupational disease or some trauma they may not be able to recover from an aggravation of their underlying cardiovascular diseases

The entrance of women in industry, begun in the early 1920's and necessarily increased by the war years, enlarges the percentage of patients with venous problems who are subject to trauma Those with arteriospasm, lymphatic blocks, and unrecognized aneurysms all add to the confused picture [4,12,20]

In certain states, *i e* , New York, the compensation claims of the patients with cardiovascular diseases or lesions provide a most complicated problem According to Dr Nicol,[11] of the 175,825 injuries reported in 1952 to the State Insurance Fund for conditions arising from trauma or the occupational hazards inherent in industry, 167 or 09 per cent were vascular lesions of such gravity to require reference to a vascular surgeon By their very nature, such problems are not of short duration, and the relative number of disability days lost is second to no other industrial lesion

Liberal interpretations of labor and compensation claims have increased tremendously the number of conditions considered related to the hazards of the patient's occupation For example, in many states it is no longer necessary for a patient to describe an injury or accident of any type in order to have a hernia declared compensable All that is necessary is that the work he performed caused strain and that in some other instance such a strain may have caused or contributed to the development of a hernia. This example carries over into the vascular field

Since patients with such lesions continue to work they must be covered from the hazards of labor by compensation insurance This does not mean that if the natural conclusions of the disease occur while a patient is at work the work is related to these conclusions For example it does not necessarily follow that merely because an arterial occlusion occurs in a patient's leg when he is at work that the work is responsible for the occlusion Occlusions in an advanced arterial lesion occur often when the patient is in bed and the time of the final thrombosis of an artery cannot be anticipated

In patients with these underlying lesions however a mild trauma may precipitate the final occlusion of an artery Such an occlusion is then related to the trauma on an aggravation basis The patient who has a diabetes mellitus may have a peripheral arterial failure following undue exposure to cold Each case must be judged on its own merits but certain fundamental principles may guide one in such rationalization Some of the principles are noted here

**The Relationship of Trauma and Occupational Diseases to Coronary Thrombosis and Myocardial Infarction.**—Patients with cardiac disease in many instances are physically capable of gainful employment Many of these are rejected from work which they can perform because of a possible claim for compensation under state laws A recent study by Goldwater's committee (supported by the New York Heart Association) is revealing in this respect[6] While the average cost per closed compensation case (340 000) in a three-year period was $455 00, the average cost of indemnity in a three-year period for cardiac patients (595) was $8 006 00 Of the controverted cases only 24 per cent were disallowed, and of those judged compensable one-third of the fatal attacks occurred when the patients were not working It is apparent from such figures that if the cardiovascular specialist's efforts to keep such patients working are to be successful, a better adjudication of these occupational episodes must occur This is further emphasized by the fact that in New York State the workmen's payment for heart disease amounted to 2 million dollars annually, which is 4 per cent of all compensation costs.[6]

There is little difficulty in connecting direct traumatic injuries and heart lesions Open wounds are caused by bullets shrapnel or stabs during war time and flying glass or metal or accidental penetrations during peace time The train of events is easily established and confirmed Indirect injuries can also damage the myocardium. A blow over the heart, a fall or jarring can cause injury to the myocardium or large vessels by rupture or by the mechanism of contra-coup

The problem is to determine which heart lesions develop after unusual or prolonged effort. It is agreed generally that the normal heart will not

be affected by extra efforts, but the effect on the diseased one may be serious To further complicate the picture, arteriosclerosis of the coronary artery can exist for years without symptoms Often, one cannot differentiate clinically between the normal and diseased cardium. The increasing incidence of this disease, which kills 200,000 persons a year and cripples innumerable others,[18] makes this an important problem While the disease has caused death at every year of life from three months to eighty years, characteristically it strikes at the most active and productive age when the individual has his greatest responsibility toward his family and his fellow-men [16] The responsibility of the doctor to determine if the patient's injury or work contribute to his disability or death is a serious one and requires knowledge, study, and above all, professional integrity Certain principles can be outlined

(*a*) It has not been proven that unusual, strenuous, or prolonged work can cause coronary thrombosis or acute occlusion While intimal hemorrhage and abscesses occur, their relationship to stress and strain has not been established without question

(*b*) Such efforts or strain can cause myocardial infarction, however, in patients with coronary arteriosclerosis

(*c*) In like manner, sudden death can occur in such individuals without myocardial infarction

These conclusions are based on the opinions of Yater,[21] Dock,[3] Blumgart[1] and Maynard,[10] and to some extent, refutes earlier opinions of Master and others [8, 9] The problem of heart disease, cardiac death and injury poses serious difficulties The number of cases which are appearing upon the Compensation Board's roll is increasing Well over 200 such cases are listed each year [6] Significantly, the number increases each time a case is decided for the patient, proving the influence of precedence on the patient and his lawyer While world changes in social and economic matters are occurring and are far-reaching, the physician's role must remain a medical one The status of the patient's home situation, his financial losses, his dependence or his "pitiful state" must not influence the doctor's opinion as to the relationship of his work and the onset or aggravation of his cardiac disease If it is the will of the majority that patients in this status shall be cared for by the state, this must be provided by legislative means and not by medical testimony More patients have a coronary attack or die of coronary occlusion in off hours than they do at work

Daily work is not a cause for coronary occlusion The number of workers who labor without developing coronary heart attacks proves this fact The ability of the patient to perform such work for years prior to his coronary occlusion is further proof that the occupation is not the cause of the lesion An unusual effort or strain, if followed by a coronary heart attack, might be considered a precipitating factor This occurs in only 2 per cent of such attacks An example could be the lifting of a heavy object by a worker whose usual occupation was desk work If the coronary thrombosis followed the lifting soon enough, one could not rule out strain as a precipitating factor

While most members of our profession are true followers of the great Healer, there are exceptions to the rule Unscrupulous doctors have been

known to assure the payment of bills for services to patients who are unable to pay by their shaded testimony in compensation court Fortunately, these doctors are few

Arteriosclerosis occurs at a much earlier age than we believed formerly Arteriosclerotic lesions have been seen in a patient aged four and a complete arteriosclerotic occlusion in the legs of a patient of seventeen During World War II 866 patients between the ages of eighteen and thirty nine died of arteriosclerotic coronary disease French and Dock[4] reported that occlusion occurred on the average of 1 per hour during sleep 7 per hour during the first two hours of the day 10 per hour during vigorous effort and 3 per hour during rest This same incidence would follow the usual exercise coincident with a vacation The ability of patients with cardiac diseases to continue their work is well-demonstrated by the fact that 8 out of 10 patients with heart disease return to their previous positions after their first attack A study of 1,840 patients with cardiac disease compared with 3 055 without heart lesions was made in 50 different industries[4a] The difference in satisfactory job performance while small was in the favor of the workers with cardiac disease

## OCCLUSIVE ARTERIAL LESIONS

A **Direct Trauma.**—Patients with occlusive arterial lesions are subject to damage from minor traumas which otherwise might not affect them Such injuries may be mechanical thermal or chemical In order that there be a causal relationship between an acute arterial occlusion and trauma one must fulfill the following conditions

1 A trauma of sufficient degree must occur to cause an injury to the artery and the trauma must not be one to which the patient usually is subject

2 The trauma should be at or contiguous to the site of the occlusion

3 The symptoms of occlusion should not have been present prior to the injury or if so these signs must be aggravated since the injury

4 The symptoms of occlusion or aggravation thereof should date from the injury or from a reasonable time thereafter

B **Indirect Trauma.**—Arterial occlusion in rare instances may result from an increased intra arterial pressure without direct trauma. An unusual or severe strain may inaugurate such an occlusion the lesion developing probably from the blood under the increased pressure undermining a thrombus or a plaque This should follow the alleged strain and not have been developing prior to this strain The history of a similar degree of effort on other occasions without occlusion would tend to rule out the work alone as the aggravating factor When a vessel is partly collapsed a milder strain may fracture the clotted or calcified area and be followed by obstruction

A precipitation or aggravation of an occlusive arterial disease in other parts of the body has been noted after a severe or continuing trauma to one part of the body For example, in one patient with thromboangiitis obliterans a trauma of sufficient degree occurred which resulted in the loss of an extremity A chain of events was inaugurated from that time on with

occlusion occurring in arteries of the other lower extremity and, later, of the upper extremities In such a case for there to be relationship between the original injury and lesions in other extremities, it is necessary for:

1 The trauma to have been severe enough to affect the artery

2 A lesion to develop thereafter at the site or near the site of injury

3 Other lesions to have developed concurrently or within a reasonable time thereafter

4 There to have been no similar lesions present in the other extremities before this injury

It is likely, in such an event, that the injury caused a migrating type of lesion in underlying diseased arteries similar to that seen in the migrating phlebitis after injury in the venous system

## SPASTIC LESIONS

This problem of industrial injuries and hazards caused by spastic lesions already has been described under such lesions as traumatic segmentary arterial spasm and Raynaud's syndrome For there to be a direct connection between the patient's work and a spastic lesion it must be demonstrated that (1) the lesion did not exist prior to the trauma or the occupational work, (2) the work in itself potentially is able to cause such spasm, (3) the lesions following the spasm developed shortly after the onset of that particular type of work An example of lesions of this type is a Raynaud's syndrome which begins shortly after exposure to cold, chemicals or after the handling of certain materials, such as sheet metal

In the use of pneumatic pressure drills and similar apparatuses there is a rush of cold air under pressure The cold air and the pressure may cause spasm An intermittent type of trauma also may cause such lesions.

Other lesions of a spastic nature may occur after exposure to such chemical irritants as glue, paints (particularly of the phosphorescent type), any of the detergent or "burning compounds," acids, alkalies, and radium, radioactive isotope and roentgen rays

Prophylaxis is the best treatment in these patients The use of resilient pads to relieve pressure or vibration and the elimination of exposure to the stimulants which produce the spasm, *i e*, glue, other chemicals, etc., are important The training of patients to do the same type of work with their left hands may help Individuals who have hazardous types of work should be trained in two different trades so they may alternate or substitute them from time to time See chapter on Spastic Disease, page 256

## DIRECT INJURIES TO THE ARTERIES

If a patient has a direct injury to his arteries at work, the relationship between the two is obvious. It is in the indirect injuries to arteries that the industrial injury problem arises, especially in the individuals already subject to some arterial disease This problem has been discussed under Occlusive Diseases and Arterial Thrombosis See pages 159, 352

## ANEURYSMS

When aneurysms are the result of a direct injury there is no question regarding the causal relationship The possibility of strain causing an aneurysm in an already diseased vessel presents a problem in the medicolegal field many times

If the blood vessel is congenitally inadequate or diseased by arteriosclerosis gout lead poisoning arteritis or syphilis it can dilate aneurysmally if an undue intra-arterial pressure is exerted An example is the patient with an underlying lesion who sits at his desk each day and performs no physical work. If he is called upon to push a heavy cart or lift a desk this strain may increase his intra-arterial pressure enough to cause an aneurysmal dilatation to occur The fact that these same lesions develop with strain or trauma makes the problem in each instance an individual and difficult one to decide Trauma may be an aggravating factor if it is of such a degree as to have possibly injured the vessel wall directly or indirectly and if this injury is at or contiguous to the site of the aneurysm

These aneurysms often rupture This is particularly true in the thin-walled vessels in the brain If cerebral hemorrhage or thrombosis occurs as the result of such rupture during the working day the possibility of the patient s work as an aggravating or exciting cause of the rupture is questioned repeatedly

Most of these diseased vessels eventually rupture If the patient s work is of his usual type and there has been no strain a rupture of the vessel even if it occurred when the patient was at work should be considered in no way related to this work The same would be true in any usual strain in the course of his employment The fact that a patient walks up and down stairs several times a day without causing any increased intra-arterial pressure would argue against one such trip being the cause of a cerebral hemorrhage if it occurred We know that rupture and hemorrhage is due to occur some day from such diseased vessels

There are many extenuating circumstances in these cases The greatest problem is presented when the aneurysmal dilation occurs with some strain but fundamentally is due to a congenital weakness or developmental abnormality Prior to birth arteries carry venous blood and vice versa. Connections between the two systems close normally before term The surprising part is not that these break open again but that they do not occur more often The breakdown in the cellular structure between the arterial and venous systems occurs most often at puberty and a few years thereafter This time corresponds with the entrance of many individuals into active and strenuous life Each case must be individualized In general however *if some* unusual and abnormal trauma or occupational strain occurred coincident with the development of the aneurysm, this injury or occupational hazard cannot be ruled out as a precipitating or aggravating cause

In these changing social times *there* will be many arguments over these cases Many lay people believe in the socialization of all work and health After a time these problems will be clarified by the will of the majority

and without question standards of health and compensation will be established We are doing our fair medical part if we say that if the patient has a disease from which eventually he may die, his work may not be related to such death, even on an aggravation basis, unless there is some unusual or unnatural strain occurring directly or indirectly

## CERVICAL RIB AND SCALENUS ANTICUS SYNDROME

As has been described on pages 470 and 478, many of these patients will go through life without any symptoms whatsoever The symptoms develop at certain times of life For example, at the time of puberty and, again, the time when there is atrophy or relaxation of the muscular development, disability may develop either from the cervical rib or from the scalenus anticus muscle hypertrophy or both

A change in occupation or vocation, as, for example, a transfer from a sedentary type to one requiring excessive straining of the neck and shoulders, may precipitate these symptoms Work which requires elevation of the arms (*i e*, painting a ceiling) may initiate the pathologic picture If the patient is not used to this type of work, there can be little question but that work was an aggravation factor If it has been shown that the patient without a cervical rib has done this same type of work for many years without any evidence of these symptoms and that other individuals with similar muscular development are doing the same type of work without symptoms, then the work can be eliminated as a cause or aggravating factor Pre-employment examinations should eliminate those with cervical ribs from doing work of heavy muscular nature in which the neck and shoulders are involved

## VARICOSE VEINS

A high percentage of patients have varicose veins after the age of forty years (79 per cent of females over forty and 41 per cent males[17]) The relationship between varicose veins and their complications and injury or occupation in industry will always be contested With simple varicose veins, direct injury may cause a hemorrhage if it lacerates the vein In such an event there is no question of causal relationship (See Etiology, Varicose Veins, page 557 )

In a patient who has advanced varicose veins certain complications will develop if the condition is not treated The veins will enlarge with a secondary dermatitis and edema, and ulceration or thrombitis develops in one-third of the patients with varicose veins

**Ulcers** —The main medicolegal problem arises in the patient with *ulcers* If a patient with varicose veins injures his leg, he may develop an open lesion which will not heal because of the underlying venous pathology

In these instances, we believe the ulcer is related to the injury on an aggravation basis This causal relationship continues until such time as the traumatic ulcer is healed and has remained healed under working conditions for a *reasonable length of time* A reasonable length of time in such cases has been accepted by most experts as from nine to twelve months

If the patient is able to work after such an injury has healed for this time without it breaking down, the trauma as an aggravating factor is no longer present. Were this trauma still a factor in the leg the ulcer would not have remained healed over that period of time.

Later it is to be expected that with this underlying venous pathology other ulcerations may occur. These are not related to the original injury provided that this reasonable length of time under working conditions has elapsed and the new breakdown is not at the site of trauma. During this period the healed lesion must have remained closed. If the lesion promptly breaks open when the patient returns to work, this breakdown then must be considered causally related again until such time as continued healing as described is present.

If another ulcer occurs at the actual site of the injury the problem of relationship is more difficult. It is true that the original injury site may be weaker for some time as the healing may have resulted in scarring. This scarred tissue is brittle and more subject to trauma. If it withstands the strain of working by remaining healed for considerable time, i.e. one year, then new breakdowns may be due to the underlying disease and not due to the trauma. One of the big problems of the relationship of ulcerations to work is the patient who has had varicose veins for many years, frequent and multiple ulcers, who then develops an ulcer which is alleged to be due to an injury or the patient's occupation. Such a patient may previously have accepted these ulcers as part of his disease process, but suddenly he believes that this ulcer and subsequent ones have been caused by his work efforts. Unfortunately this may be abetted by unscrupulous legal and medical advice and testimony. The doctor thus is placed in the dual role of medical and legal assistant, and since his decision may be far reaching in its economic effect, his responsibility of evaluation becomes greater. The problem presented by the dishonest patient who obtains medical and surgical aid for a chronic unaggravated condition is always present. The doctor must have this possibility in mind at all times. Each case must be individualized. To some extent such patients present a problem to social medicine and not industrial medicine. This economic problem so far is not solved.

## THROMBITIS (THROMBOPHLEBITIS, PHLEBITIS)

By thrombitis is meant an inflammatory condition in a vein or veins with a secondary clotting. Such inflammation may be manifested by a local or streak-like redness. Usually there is venous stasis and congestion distal to the process.

There are two types of thrombitis. The patient who injures a normal vein, in which a clot and/or inflammation develops, has a venous stasis due to this obstruction. Such venous pathology causes dilated veins which act as collateral vessels and the inflammation may spread to other previously uninvolved veins. The other type of thrombitis occurs in the patient who has varicose veins or other venous pathology and secondarily injures the veins. In this latter type the traumatic aggravation must be considered to continue until the status quo ante is reached or an end stage

of improvement occurs In the former type, the status quo ante may never be reached because of secondary permanent pathologic vein changes These may be manifested as enlarged veins, edema, skin changes and recurrent attacks of inflammation Ulcers are a complication in this picture

The time schedule for causal relationship to cease must be changed if thrombitis is present These problems of the relation between simple varicose veins and ulcers are not too difficult to interpret if the underlying principles are kept in mind

When the injury causes a thrombitis (*thrombophlebitis*), however, then the problem is a much more serious one The thrombitis, once set up, may continue in a chronic, subacute, or low-grade state over a long period of time and be subject to recurrent symptoms The pathologic picture in varicosities due to thrombitis is entirely different than that due to varicose veins It is more difficult to decide when the lesion is healed permanently Each case must be individualized

In general, if the thrombitis is quiescent for a reasonable length of time, for example, a year, the aggravating factor may be considered terminated If, on the other hand, there are secondary changes from the phlebitis such as edema, ulcerations, and scarring, these may be considered to be related on an aggravation basis permanently, and reactivation, even in a slightly longer time than one year, in the individual instance, might be considered related See page 616

These cases must all be considered separately The status prior to the injury must be established Pre-employment examinations are of utmost importance to register prior vein inflammations or lesions

## THROMBOSIS

Venous thrombosis means, primarily, clotting in one or more veins At an early stage, inflammation is not present, and in some patients it never develops If the clotting continues long enough, inflammation will develop It is from this type of vein that embolism most often occurs

Thrombosis of one of the veins may be related to a patient's occupation if there is a direct injury In direct trauma there will be no question of causal relationship Sufficient pressure on the vein wall for a considerable length of time may cause injury to the endothelium, stasis and then thrombosis Stasis alone may cause thrombosis

Because of the extensive pathology and thromboembolism, the causal relationship factor becomes of utmost importance The history must be correct The status of the venous system prior to the alleged trauma or occupational hazard also is important An important cause for thrombosis is *bed rest*, and therefore someone who is leading an active life is less likely to develop thrombosis Thus, work *per se*, unless of an unusual type or requiring the patient to be in a peculiar position, is not a likely cause for venous thrombosis

The postoperative thrombosis and its complications if they occur after an operation for a compensable injury, *i e*, a hernia, are related The complications of the thrombosis, such as embolism, thrombitis, ulcer and re-

currence of the process must be indirectly related to the original venous insult.

Any such short discussion of vein problems and trauma or occupation necessarily is superficial and incomplete. This subject constitutes one of the most difficult sides of compensation and insurance medicine and only broad generalized statements can be made. It is understandable for two qualified ethical men to have divergent opinions on the relationship of injury and disease in the same patient. Such controversial thoughts rarely cause the problem and usually can be resolved. It is the positive statement of the compensation wise but medically dishonest physician which causes most of the insurance troubles. The feeling that the individual is always entitled to everything he can get is valid only if the facts substantiate his claim.

No one today opposes the provision of medical care to a worker for any legitimate injuries or occupational hazards. It must be understood however that people develop diseases as they grow older despite work. More older people are working today than formerly.

Some doctors and jurists wish to interpret compensation and disability laws to cover all complications of all diseases. Some will relate a pneumonia to a draft in the office. Others interpret the laws to cover the termination of life in the cardiac to the patient's work if death happens to occur during working hours. If this trend were carried one step further to include the worker's family, automatic socialization of medicine will have arrived. These are political not medical problems. If the majority of the people wish it they will enact insurance laws to cover these degenerative diseases. That must be done by political votes and not by medical interpretation of existing insurance laws. If the employers are held responsible for all complications of degenerating diseases they soon will refuse to employ any but the young and most healthy individuals. This will be a disservice to the many older people who still can work, need the work and whose experience is valuable.

The tendency to keep people working many years longer than in past years with the help of good medical care makes it important that certain standards be set up so that the new employer will not be held financially liable all of his life for the patient's degeneration due to his underlying disease. The Second Injury Law of New York State is an effort in this direction.[6]

In summary the relationship between work and injury in a direct injury is based on fact. The relationship between an indirect injury and a vascular lesion depends on fact and the following:

*a* that the indirect injury was a competent cause to produce the lesion

*b* that the lesion did not exist before the injury

*c* that the lesion began at the time of or shortly after the injury

The aggravation of an underlying disease is possible if the conditions elaborated under indirect injury are fulfilled.

The status quo ante in an injury or disease of a vascular type can be considered to be reached when the patient has been in the same status as he was prior to the injury and remained so under working conditions for a reasonable time.

Occupations vary as to their hazards to health Each case must be considered separately and decided on its merits No generalized rules can be established to guide the decision as to causal relationship Certain points should be considered (*a*) the effect of this work in general on all workers, (*b*) the effect of this work in the past on this particular individual, (*c*) the status of this individual's cardiovascular system, and (*d*) any variation from the normal in this patient's work

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